VOLUME 42 NUMBER 3

ARCHIVES OF

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374

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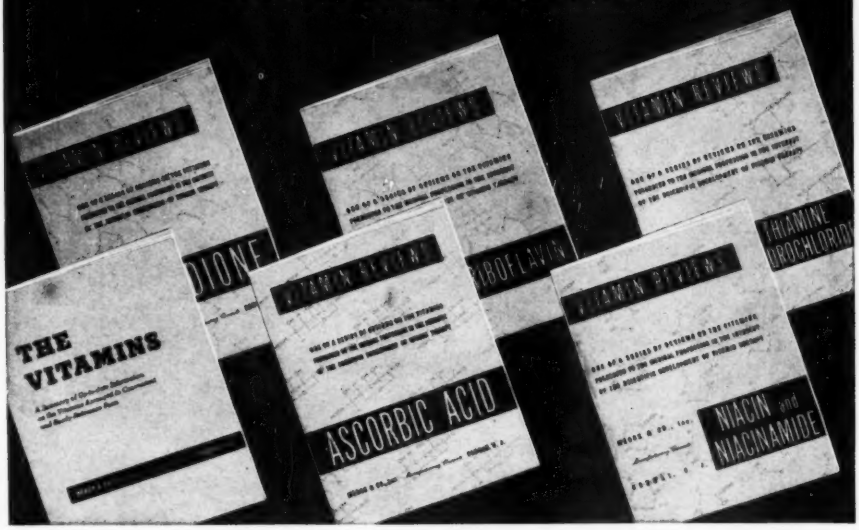

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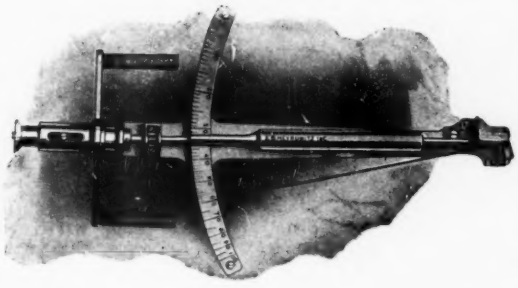

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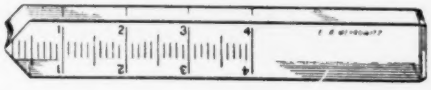

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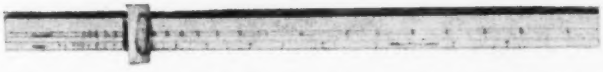

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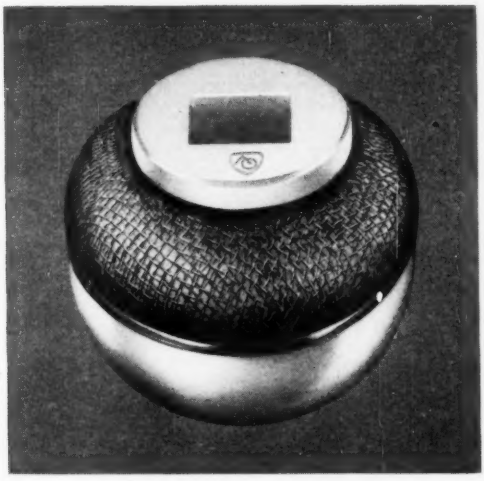

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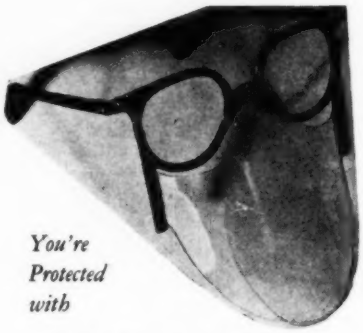

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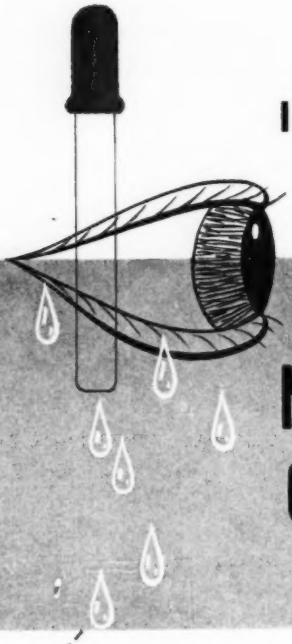

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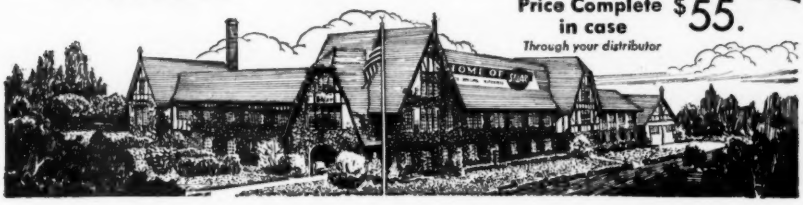

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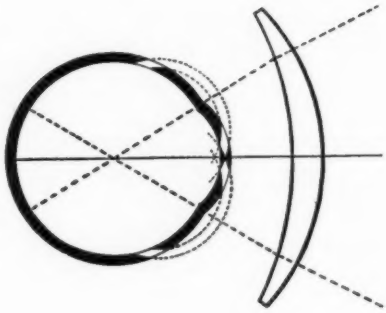

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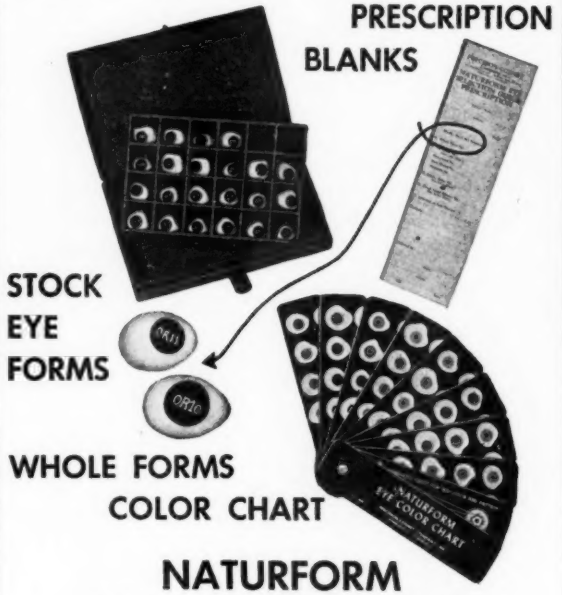

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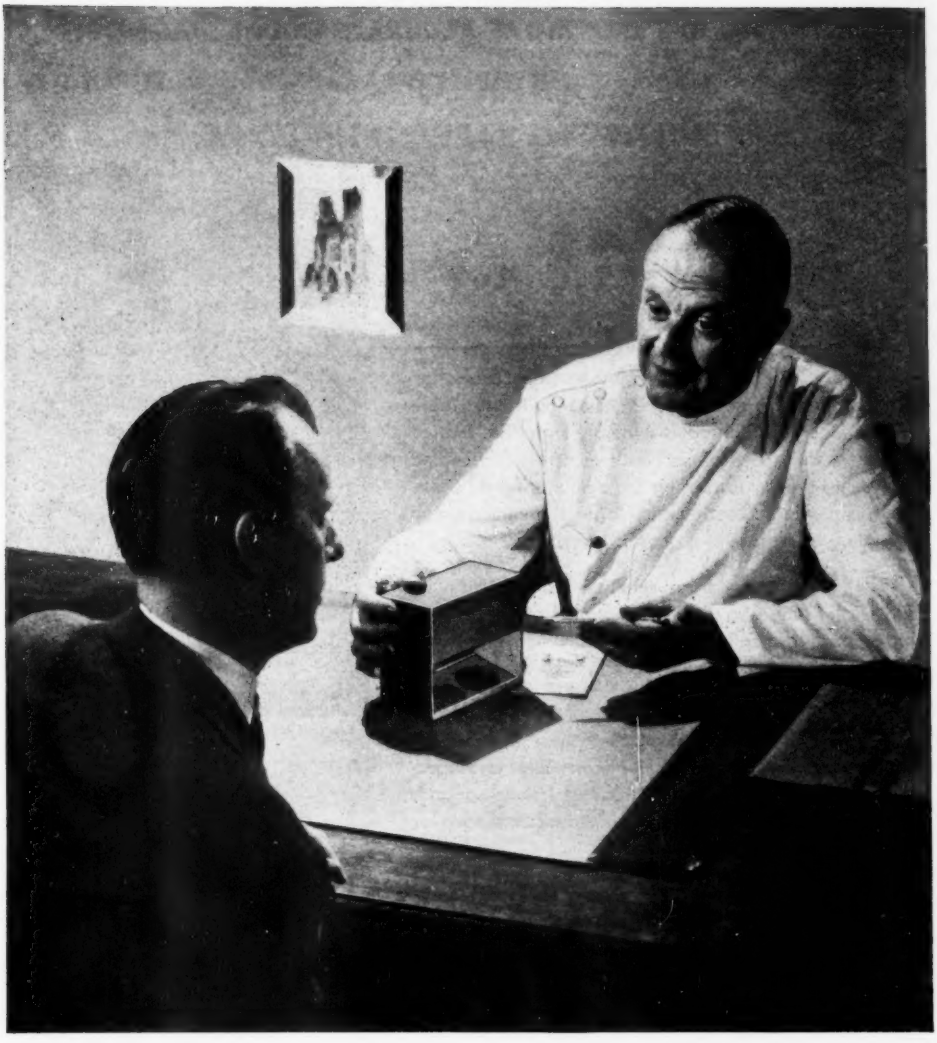

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ARCHIVES OF OPHTHALMOLOGY

VOLUME 42 SEPTEMBER 1949 NUMBER 3

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HEREDITARY MYOPIA

KARL C. WOLD, M.D.
ST. PAUL

THE THEORIES concerning the etiology of myopia are many and varied and have undergone a change as time has gone on. Von Graefe suggested that myopia was caused by subacute inflammation of the choroid and sclera. This theory has been disproved microscopically by the fact that the hyperemia, exudation and cicatrization found in myopia are only processes of repair which follow stretching and lacerations of ocular tunics. In 1610 Kepler stated the belief that the influence of near work, which requires accommodation and convergence, was an etiologic factor. Donders disproved this basic principle by showing that the highest grades of myopia may be found in illiterate persons and in people who never engage in close work. This theory was also disproved by Holm,[1] who made a thorough study of ocular refractive errors in the natives of Gaboon, French Equatorial Africa, one of the most ancient strains of the Negro race. These people showed tendencies in ocular refraction, from childhood to old age, more or less parallel with those found in the civilized world. The refractive variations were less conspicuous than among civilized communities, but this observation still bears out the theory that heredity plays the main role in myopia. Crisp concluded that three factors are concerned in the etiology of myopia, namely, (1) heredity, (2) nutrition and (3) the influence of study and close work, and that they may singly or jointly be responsible for the condition. This is a reasonable theory except that the second and third factors should be classified as contributory only.

Gates [2] gave the following source material on the etiology of myopia: Clarke (1925) suggested that myopia comes from stretching of fibroblasts in very early embryonic growth. He estimated that one child in a thousand is myopic by the sixth year but that at the age of 20 years 15 per cent are myopic. Parsons regarded congenital weakness of the sclera as hereditary and therefore a distinct factor in myopia.

Read at a meeting of the Minnesota Academy of Ophthalmology and Otolaryngology at St. Paul, Jan. 16, 1948.

1. Holm, S.: Heredity in Myopia, Am. J. Ophth. **21**:680, 1938.

2. Gates, R. R.: Human Genetics, New York, The Macmillan Company, 1946, vol. 1, p. 192; vol. 2, p. 934.

Watson (1934) found a pair of twins both of whom were myopic and in both of whom diabetes mellitus developed at 56 years of age. This finding is similar to a case in my experience (kindred 257), in which both mother and daughter had onset of diabetes and myopia at the age of 49.

Steiger's [3] article on the etiology of myopia is concerned with a controversy between H. Becker, of Munich, and himself (Zurich). At that time there were two prevalent theories about myopia: (1) the Lamarckistic, and (2) the Darwinistic. The Lamarckistic theory was based on the concept that myopia is simply an adaptation of the eye to present civilization. The Darwinistic theory assumed that the myopic eye is congenital and nothing can be done about it. Becker expressed the belief that myopia can be brought to a standstill by early correction, especially of the astigmatism, which is often only —0.25 or —0.50 D. Steiger adhered to the Darwinistic theory and stated the belief that correction of the refractive error, even in early childhood, will not prevent the progress of myopia.

Lindner [4] expressed the belief that myopia is not congenital but that the intensive use of the eyes, especially during the developmental years, has a great deal to do with its development. He stated the opinion that the rapid progress of myopia can be prevented by proper correction in early use. His theory of the causation of myopia is that there is a certain weakness of the sclera in the posterior segment of the eye near the papillary area. This weakness is caused by a change in the capillary blood vessels, due to constant edema in that region, which, in turn, is produced by a constant abuse of the visual organs in doing continuous close work.

Grunert [5] made the following observations on myopia: It is inherited as a normal variant in a biologic sense. It is a symptom of civilization, because in the domesticated being the extreme variants are not eliminated in the fight for survival but are preserved and propagated. He also claimed that there is a "physiologic inferiority" in the posterior portion of the eyeball in childhood. Grunert summarized his paper by saying that the hereditary tendency to *Dehnsucht* (stretching of the eyeball) in myopia consists mainly in the inheritance of an insufficient ciliary muscle and a particular weakness of the connective tissue of the sclera.

3. Steiger, A.: Einige Bemerkungen zu Dr. Deckers Aufsatz: Klinische Beobachtungen über die Ursache der Kurzsichtigkeit, Berl. klin. Wchnschr. **56:** 419, 1919.

4. Lindner, K.: Neue Gedanken über die Entstehung der Kurzsichtigkeit, Klin. Monatsbl. f. Augenh. **103:**583, 1939.

5. van Grunert, Karl: Die Dehnsucht des Auges (Myopie) und ihre Behandlung, München, J. F. Lehmanns, 1934, pp. 8, 12, 16, 17 and 158.

The theory of compression of the globe by the extraocular muscles was advanced by von Arlt,[6] and later by Stilling.[7] It is possible that muscular contraction could injure an elongated globe but could not itself change the form of a normal globe with ordinary elasticity.

Hassner, and later Weiss,[8] decided that an optic nerve which is too short could pull on the back of the eye when the anterior part of the globe is turned in convergence. This traction would have the effect of producing a posterior staphyloma and consequent myopia. There has never been anatomic proof of this theory.

Stocker[9] stated that retinal changes, such as atrophy of the pigment epithelium, rods and cones and cystic degeneration, are determining factors in producing the stretching process, rather than changes in the choroid or the sclera. Heredity would still be the cause of myopia even though this theory were true.

All have seen cases in which myopia developed at 6, 10 to 12 or 18 to 20 years of age, roughly speaking, and have wanted to blame the influence of close work, greatly increased body growth or changes in calcium and thyroid metabolism. These cases reveal the hereditary pattern of myopia and show the influence of contributing factors only. The following conditions are the most probable contributing factors in the development of myopia: (*a*) Convergence. The action of the ciliary muscle in contraction pulls the choroid forward and breaks its insertions at the disk (as in myopic crescent). Binocular fixation necessitates rotation of the eyes inward, which is all the more pronounced as the point of fixation is nearer. The tighter this belt of internal and external rectus muscles becomes, the more elongation of the globe takes place, and the band might well interfere with the circulation in the vorticose veins and thus cause congestion.

(b) Accommodation. The concomitant effect of accommodation, though not greatly exerted in a myopic person, may cause peripapillary alterations of the choroid, due to traction exerted on it by the longitudinal fibers of the ciliary muscle (tensor of the choroid), resulting in venous congestion of the uveal tract, a condition most favorable to permanent alteration of the deep membrane.

(*c*) Close work and reading. The bending of the head to such work, with the subject usually in a sitting position, and the corre-

6. von Arlt, C. F.: Ueber die Ursachen und die Entstehung der Kurzsichtigkeit, Vienna, Wilhelm Braunmüller, 1876.

7. Stilling, J.: Ueber das Wachsthum der Orbita und dessen Beziehungen zur Refraction, Arch. J. Augenh. **22**:47-60, 1891.

8. Weiss, L.: Beiträge zur Entwicklung der Myopie, Arch. f. Ophth. **22** (pt. 3): 1-124, 1876.

9. Stocker, F. W.: Pathology and Anatomy of the Myopic Eye, with Regard to Newer Theories of Etiology and Pathogenesis of Myopia, Arch. Ophth. **30**:476 (Oct.) 1943.

sponding sedentary state both contribute to congestion in the head, especially the eyes. This is especially true when the visual acuity is poor and an attempt is made to magnify objects by getting closer to them. Poor illumination also contributes to this factor.

(*d*) Abnormal function of calcium and thyroid metabolism. After considering all these opinions, theories and facts, one must conclude that myopia is caused by a congenital defect which exists in the resisting power of the sclera during the period the growth of the anteroposterior diameter of the eyeball. This defect is passed on through inheritance and is influenced by the aforementioned contributing factors.

TYPES OF MYOPIA

A. Anatomic Types: 1. Axial. Long eyeball with focus of parallel rays of light in front of retina.

2. Abnormal refraction. Excessive curvature of cornea or lens capsule.

B. Etiologic Types: 1. Acquired myopia. This type is found as a result of disease and trauma. Predisposing conditions are (*a*) changes in lens fibers associated with diabetes and intumescent cataract; (*b*) anterior staphyloma, caused by injury or disease of the cornea; (*c*) choroiditis and sequelae; (*d*) spasm of accommodation; (*e*) change in refractive index of the intraocular fluids, and (*f*) anterior displacement of the lens.

Weekers [10] reported the case of an 11 year old boy who was struck forcibly in the left eye. A —3.50 D. sphere brought normal vision to this eye in twenty-four hours, and in one month a + 1.00 D. sphere was needed. The intraocular tension was very low as determined by palpation when the myopia developed. This transient myopia was attributed to relaxation of the zonule brought about by hyperemia and edema of the ciliary body, and the lowered tension, to absorption of the aqueous. Transient myopia also comes from chemical changes in the lens fibers, as after administration of the sulfonamide drugs and dinitrophenol. Acquired myopia constitutes less than 1 per cent of all types.

2. Inherited myopia. The genetic theory of myopic inheritance recognizes that each nucleus of the human ovum and sperm cell contains twenty-four paired chromosomes. The determining factors or genes are assumed to exist within the chromosomes like beads on a string. These genes go to make up the pattern of inheritance, which is classified as three types:

(*a*) A dominant character is one possessed by one of two parents of hybrids which appears in the hybrids to the apparent exclusion of the

10. Weekers, R.: Ann. d'ocul. **178**:236, 1945.

antagonistic character. It is usually designated by a capital letter, such as B. A parent with a dominant factor will pass it on to 1 in 2 children, regardless of sex.

(*b*) A recessive character is one which is apparently not inherited by the hybrids, but is nevertheless latent and appears in their descendants, regardless of sex. For a child to have a recessive condition, each parent must contribute the same gene, which is designated by a small letter, such as *b*.

(*c*) A sex-linked recessive character is one which is carried from one sex through the second generation of the opposite sex to appear in the third generation of the original sex. This character is commoner in males than in females and is not passed on by affected males to their children, but their daughters transmit the character to half their sons. Myopia is one of the apparent thirty sex-linked traits in man.

TABLE 1.—*Incidence of Myopia in 231 Children Under 6 Years of Age*

Age Group	Myopia	Hypermetropia	Normal
Premature	..	5	..
1½ hours to 7 days	2 (2.7%)	55	11
7 days to 1 year	1	9	8
1 to 2 years	1	12	..
2 to 3 years	1	20	1
3 to 4 years	4	21	1
4 to 5 years	3	33	1
5 to 6 years	3	35	4
Totals	15 (6.5%)	190	26

Stansbury [11] classified myopia into two groups: (1) primary myopia, which he termed a biologic variation, with clinical evidence that it is a healthy refractive state, and (2) secondary myopia, caused by disease of the eye in which a low grade chronic choroiditis is the primary cause.

INCIDENCE OF MYOPIA IN CHILDREN UNDER 6 YEARS OF AGE

According to Fuchs, about 5 per cent of infants are born myopic; 15 per cent are emmetropic, and the rest have a hypermetropia of 0.50 to 4.00 D.

Public health surveys in the United States and Great Britain have shown that the percentage of myopia increases from 1.1 per cent, at 2 years of age, to 10 per cent in Great Britain, but only to 7.8 per cent in the United States, by the twelfth year, the latter figure not including data on the newborn.

In a survey recently conducted by the Better Vision Institute, it was found that 7 per cent of all American children entering kinder-

11. Stansbury, F. C.: Pathogenesis of Myopia, Arch. Ophth. **39**:273-299 (March) 1948.

garten are myopic, that 24 per cent are myopic on graduation from high school and that 31 per cent are myopic on graduation from college.

A personal review of all children seen during the last year who were under 6 years of age, including 73 newborn infants at the hospital nurseries, is presented in table 1. Each newborn baby was wrapped in a blanket and taken to a dark room. The nurse then held the lids open and the baby's eyes would almost immediately fix on the retinoscope light. Few of the pupils contracted even halfway, and the movement of the shadow was easily seen. Occasionally the pupils contracted moderately, but a satisfactory reflex was always obtained. The 5 pre-

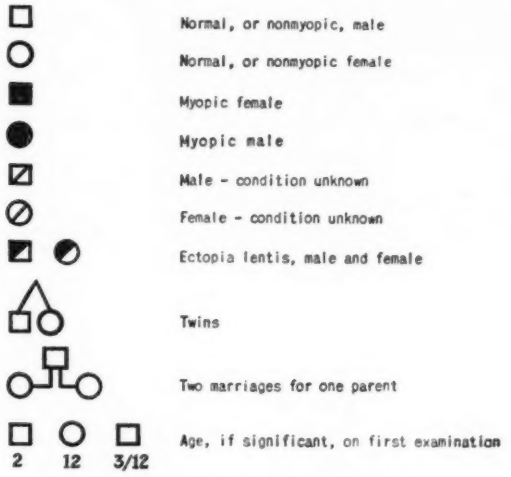

Chart 1.—Symbols used in charts 2 to 6.

mature babies, who were all hypermetropic, varied in gestation age from 6½ to 8 months. Two myopic infants were found among the 73 newborn infants, an incidence of 2.7 per cent. Fifteen of 231 children under the age of 6 years were myopic (6.5 per cent). Two sets of twins less than 7 days old were hypermetropic. One infant with myopia at birth had myopic parents, the father wearing a —0.75 D. sphere and the mother wearing a —4.75 D. sphere.

A careful study has been made of 258 kindred from my personal files in which at least two generations of proved myopia or of myopia in different and related families of the same generation were found. I have selected a few of the more interesting types, with explanatory notes for each kindred.

REPORT ON THIRTEEN KINDRED

KINDRED 2 (chart 2*A*).—1. The trait showed sex-linked inheritance from the first to the third generation.

2. The pattern of dominance appeared for five generations in one branch.

3. In the fifth generation 1 male was myopic at birth.

4. In one branch of the third generation 10 nonmyopic members failed to alter the pattern of dominance in the fourth generation.

5. In the second generation 3 males died in young manhood of pulmonary tuberculosis, but none wore glasses.

KINDRED 35 (chart 2*B*).—This kindred presents a perfect example of sex-linked inheritance.

KINDRED 65 (chart 3*A*).—This pedigree is an unusual instance of sex-linked red-green color blindness with dominant myopia.

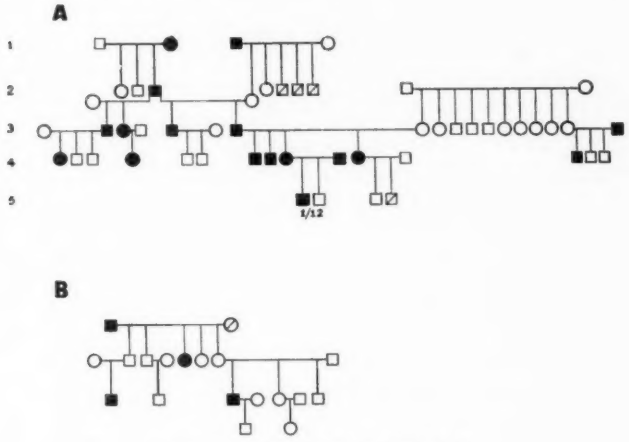

Chart 2.—*A*, kindred *2*; *B*, kindred 35.

KINDRED 87 (chart 3*B*).—1. The unmarried twin has severe myopia; she wore glasses in the first grade and throughout school life sat in the front of the room. Her baby pictures show widely dilated pupils, and the general history indicates that she was myopic at birth.

2. The married twin has much less myopia but has carried the strain through to her son.

3. Although these twins are not identical, they have inherited myopia of different degrees.

KINDRED 122 (chart 3*C*).—This kindred illustrates three definite patterns of inheritance: (1) myopia transmitted as a dominant, in this kindred from the second to the third generation, and possibly to the fourth generation; (2) retinitis pigmentosa, inherited as a dominant in various stages; (3) color blindness, in 2 siblings, transmitted as a sex-linked character through the mother on the opposite side of the family.

KINDRED 129 (chart 4 *A*).—1. All the myopia except that in the fourth generation was very severe, —6 to —11 D.

2. Most members of the fourth generation are under 5 years of age and are potentially myopic.

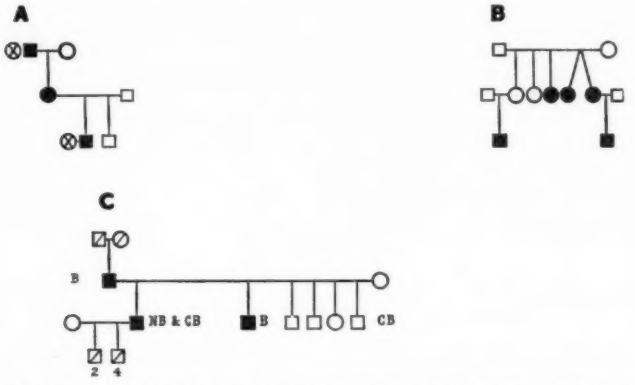

Chart 3.—*A*, kindred 65, with associated red-green color blindness (circle with cross); *B*, kindred 87; *C*, kindred 122. In *C*, *NB* indicates night blindness with early stage of retinitis pigmentosa; *B*, terminal stage of retinitis pigmentosa with retinal atrophy, and *CB*, total color blindness.

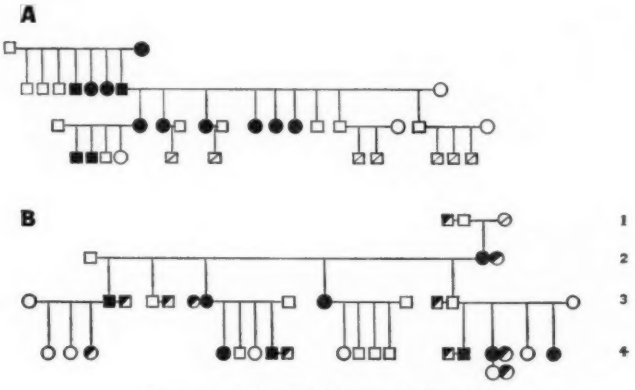

Chart 4.—*A*, kindred 129; *B*, kindred 136.

3. All 6 E. sisters in the third generation are myopic, and all 3 E. brothers in the same generation are nonmyopic.

4. This history shows typical dominant myopia and is unusual because of the severity of the myopia in each patient.

KINDRED 136 (chart 4*B*).—1. Members of the family with ectopia lentis were myopic or nonmyopic, depending on whether they were looking through the lens or through the aphakic portion of the pupil.

2. This pattern shows true dominance for both myopia and ectopia lentis, with proved cases.

3. This kindred is a proved instance of inherited lenticular myopia, because the corrections for all myopic members were —7.00 to —10.00 D. sph. when they were looking through their lenses and from +8.0 to +13.00 D. sph. when they were looking through the aphakic portion of the pupil.

4. All myopic patients had either divergent strabismus or a high degree of exophoria.

The most interesting member in this family was T. C., in whom a transition from myopia to hypermetropia occurred. A brief of her case history follows.

Aug. 9, 1945: Refraction after mydriasis:

O.D.: —7.50 sph.
O.S.: —4.50 sph. ⌒ —1.50 cyl., axis 60

The patient was very comfortable until Feb. 1, 1947, when vision became blurred for both distance and close work.

July 15, 1947: Retinoscopic study with a mydriatic was done; the correction was as follows:

(*a*) Through lenses:
O.D.: —5.00 sph.
O.S.: —7.00 sph. ⌒ —1.50 cyl., axis 30

(*b*) Through aphakic portion:
O.D.: +10.00 sph. ⌒ +2.50 cyl., axis 105
O.S.: +8.00 sph. ⌒ +2.00 cyl., axis 75

On the same date a subjective test gave the following correction:

(*a*) Through lenses:
O.D.: —10.00 sph.; 6/60
O.S.: —7.00 sph.; 6/30

(*b*) Through aphakic portion of lenses:
O.D.: +13.00 sph.; 6/15 +2
O.S.: +13.50 sph.; 6/15 +2

July 16, 1947: Because plus lenses gave better vision, the following correction was prescribed:

O.D.: +12.50 sph.; 6/15 +2
O.S.: +13.50 sph.; 6/12 +2

The patient looked through the center of these lenses, while with the old minus lenses she was forced to turn the head to one side and look through the margin of the crystalline lenses in order to get clearer vision.

Aug. 20, 1947: Lenses prescribed in July caused occasional nausea and discomfort; so with the opening of school the following prescription was given:

O.D.: +12.50 sph.; with +2.50 sph. added
O.S.: +13.50 sph.

Oct. 10, 1947: The patient had been assigned to a sight-saving class but preferred regular class and was completely comfortable with bifocal lenses.

KINDRED 163 (chart 5 *A*).—1. A myopic female was married twice, each time to a nonmyopic male.

2. Dominance is more prominent on one side of the family because of recessive genes in the male on that side.

KINDRED 174 (chart 5 *B*).—1. Myopia is recessive except for the last family in the second and third generations.

2. Despite myopia in the female of the first generation, all evidence on the unknown first generation side points to the recessive inheritance.

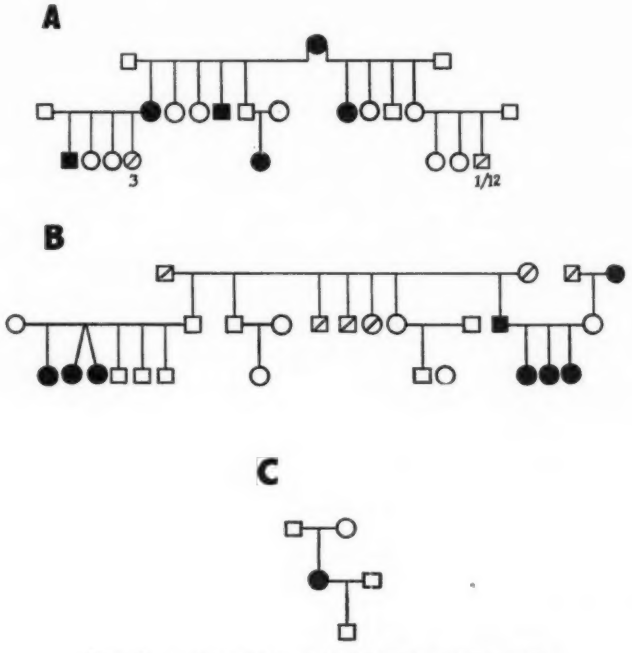

Chart 5.—*A*, kindred 163; *B*, kindred 174; *C*, kindred 256.

3. Identical twins in the third generation have myopia, although in one twin it is moderate and in the other severe.

4. The myopic sister of twins had scarlet fever at the age of 7; is subnormal mentally and had a glandular disturbance, the significance of which is doubtful.

KINDRED 256 (chart 5 *C*).—1. There was no history of myopia in the family.

2. In January 1940 the affected member was given a correction of +5.00 sph. for each eye, with +1.00 sph. added.

3. For eight months in 1940 he had recurrent herpetic ulcers of the left cornea and was hospitalized three times.

4 November 1947: The following correction was given:

O.D.: +5.00 sph., with +1.75 sph. for reading

O.S.: —1.75 sph.

5. This patient has a typical case of acquired myopia from corneal disease, a not infrequent occurrence.

KINDRED 253 (chart 6 *A*).—1. The unknowns in the first generation undoubtedly produced a dominant strain, extending to the fourth, and possibly to the fifth generation.

2. When a female in the third generation married a male with decidedly no tendency to myopia, 2 of 4 offspring were still myopic.

3. In the fourth generation the myopic female to the right has inherited the trait as a recessive, through the mother, whose antecedents were unknown.

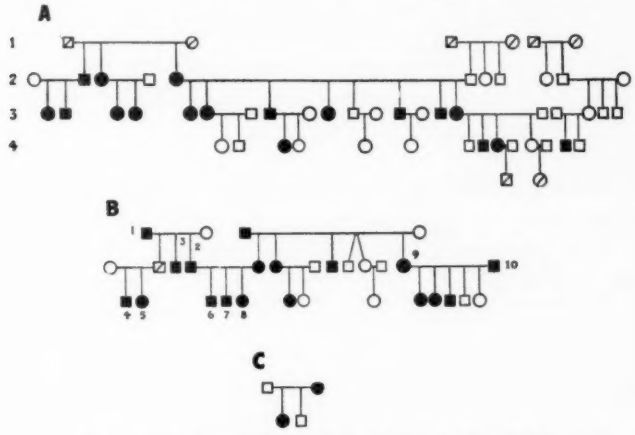

Chart 6.—*A*, kindred 253; *B*, kindred 255; *C*, kindred 257.

KINDRED 255 (chart 6 *B*).—1. Severe myopia was found in *1*, a man who also had incipient cataract in each eye before death.

2. *2*, who was a minister, had mild myopia of less than 1 D.

3. *3* the monocular diplopia, due to changes in the lens, and the myopia showed the same incidence as in his father.

4. Correction of —5.00 sph. was given for each eye. Myopia developed early in this boy (*4*). Correction for the sister (*5*) was —6.00 sph. for each eye. This girl's myopia was similar to her brother's.

5. These three siblings *(6, 7* and *8)* show how the inheritance of myopia varies in the same family. (*6*) Correction was 12.00 sph. for the right eye and —13.00 sph. for the left eye. (*7*) Correction was —5.50 sph. for each eye. *8* had mild myopia.

6. Correction was —8.00 sph. for the right eye and —9.00 sph. for the left eye.

7. Moderate myopia in *10* and rather severe myopia in his wife resulted in myopia in 3 of his 5 children.

8. Two affected males in the first generation produced the dominant pattern through three generations.

9. Only the twins in the second generation show the recessive trait, with the condition of one unknown.

10. Dominance is definitely shown in the union of two main families.

KINDRED 257 (chart 6*C*).—Mrs. M., aged 49, shows an interesting hereditary history of myopia. Her vision became blurred three years ago and had been much worse during the last year. At the age of 25, she wore a correction of —0.62 sph.

TABLE 2.—*Comparative Data in Incidence of Myopia in Children of Myopic and Nonmyopic Parents*

A. Neither parent myopic	
Males	
65 myopic	Purely recessive
201 nonmyopic	230 myopic
Females	415 nonmyopic
165 myopic	35% myopic
214 nonmyopic	
B. One parent myopic	
Males	
124 myopic	
180 nonmyopic	
Females	
185 myopic	
139 nonmyopic	Dominant
C. Both parents myopic	309 myopic
Males	319 nonmyopic
11 myopic	49% myopic
3 nonmyopic	
Females	
9 myopic	
4 nonmyopic	

for each eye but had not worn these glases for the last two or three years. At the age of 49, twenty-five years later, she was given the following correction:

O.D.: —3.00 sph. ⊃ —1.50 cyl., axis 90

O.S.: —4.00 sph. ⊃ —1.00 D. cyl., axis 90

In addition to the myopia, examination revealed diabetes, which was well controlled by insulin, and radial opacities in each lens.

The patient's mother had gone through the same sequence at the same ages, and she eventually had had both cataracts extracted.

This case presents an exception to disease as the cause of acquired myopia in that there was a definite pattern of inheritance of both myopia and diabetes.

For comparative study a tabulation was made of children who had nonmyopic parents but who were included in the kindred with myopia (table 2). Similarly, those with myopia in one or both parents were studied and classified.

TREATMENT OF MYOPIA

A careful and thorough refraction, with consideration given to accommodative power, muscle balance, age and occupation, is the best treatment for hereditary myopia. Proper light, position of the head in relation to reading material, outdoor recreation, well balanced diet and choice of occupation are important factors. All treatment such as stimulation of the ciliary muscle, administration of calcium for the sclera and thyroid for correction of low metabolic rate and medication for choroiditis cannot alter the hereditary pattern and is therefore futile.

Treatment for acquired myopia includes use of ordinary or contact lenses, cataract extraction, penicillin therapy, local treatment of the eyes and insulin, as indicated. Even in cases of hereditary excessive curvature of the cornea, contact lenses will reduce the amount of myopia and often eliminate astigmatism.

SUMMARY AND CONCLUSIONS

There are two types of myopia: (1) hereditary, which may be dominant, recessive or sex linked, and (2) acquired, due to disease or trauma.

Dominant myopia is the most prevalent type, followed closely in frequency by the recessive type, the sex-linked recessive form being the least common (table 2).

Myopia was found in 2.7 per cent of newborn infants under 7 days of age and in 6.5 per cent of all children up to 6 years of age.

In a survey of 257 kindred with myopia, the following data were obtained:

1. With neither parent myopic, 230 of 645 children, or 35 per cent, were myopic. In these children the myopia was purely recessive.

2. With one or both parents myopic, 309 of 628 children, or 49 per cent, were myopic. In these children the trait was dominant.

OCULAR ALLERGY

Allergic Phenomena Affecting the Eye and Its Adnexa

WILLIAM D. GILL, M.D.
SAN ANTONIO, TEXAS

THE OPHTHALMOLOGIC aspects of allergy have occupied a prominent place in the development of knowledge of the subject from the beginning. A number of widely different ocular disorders, both external and internal, are of allergic origin. Dermatitis of the eyelids and conjunctival edema, vernal conjunctivitis, conjunctival folliculosis, subconjunctival hemorrhage, retinal hemorrhage and optic neuritis may be mentioned as ophthalmic conditions that are often due to allergy. Substances to which a person is specifically sensitized, such as plants and their pollens, cosmetics, nail polish, soaps, shampoos, drugs, hair dyes and emanations from fur, feathers and animals, are a few of the great variety of agents which may produce contact dermatitis. Lacquers, paints, paint solvents, refrigerants and other volatile substances used in industry are also frequent offenders. Aside from the local reaction which such agents may provoke, they may, by oft repeated inhalation, give rise to a variety of internal disorders, such as optic neuritis, ocular palsies and paralyses due to lesions produced in the central nervous system. The effects of the reaction-producing substances, or allergins, vary with the individual patient, some persons being more easily affected by them than others. The sulfonamide drugs, as well as penicillin, introduced into the conjunctival cul-de-sac produce reactions in a high percentage of patients, the incidence of such reactions being so great that some ophthalmologists have discontinued their local use in the eye altogether.

Reactions may occur in the eyelids or conjunctiva as an associated manifestation of allergic rhinitis, or hay fever; the red, engorged conjunctiva and swollen eyelids being a familiar part of the nasal syndrome. Protein substances and toxins generated in the growth of bacteria and pathogenic molds or their spores are generally accepted as being capable of producing allergic reactions in the skin of the eyelids or in the conjunctiva. Sometimes the reaction produced in the eyes from contact with commonplace substances may be so great as to

Read at the annual meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy, Chicago, Oct. 9, 1948.

constitute a serious economic handicap. I have personally observed 3 persons in the bakery business who were so intensely sensitive to wheat products (flour) that their continuance in this occupation was impractical, because of the persistent reactions in their eyes. Instances of severe local reactions from atropine and homatropine have come under the observation of almost every ophthalmologist. When such reactions occur, they usually come on within a few minutes after the introduction of the drug into the eye, the reaction being so severe in some instances the eyes may be closed by swelling within a few minutes. Vesiculation in the skin of the eyelids tends to occur when the reaction is slow in developing, this being most likely to occur if repeated consecutive applications of the offending drug are persisted in, and if its use is long continued a chronic eczematoid state may be produced in which there is marked serous exudation. In postoperative care of cataract patients, in which atropine is commonly employed, the appearance of irritation and persistent swelling of the eyelids as a prominent feature should suggest sensitization to atropine. Persistent, unexplained conjunctival irritation may be due to sensitization to some particular drug used daily in or about the eyes. Physostigmine and pilocarpine drops, commonly employed in the treatment of glaucoma, often produce vascular reactions in the eye, especially after use for several weeks. This type of reaction is considered by most authorities as a toxic, rather than an allergic, reaction. Almost without exception, patients with glaucoma reach a point where physostigmine produces local irritation in the eye and must be discontinued. Pilocarpine produces a similar reaction, but when the patient acquires sensitivity to one of these drugs a change from one to the other may be made without perpetuating the irritation. Therefore, the simultaneous use of these drugs as eye drops, which is sometimes advised, is open to a degree of objection. Carbachol, a new drug used in the treatment of glaucoma, may be a valuable substitute in cases in which both physostigmine and pilocarpine have induced local reactions, but carbachol, like physostigmine and pilocarpine, may produce local reactions in the eye. A change in the p_H of the tears is apparently associated with the production of such reactions. The higher the p_H the more likely is the reaction to develop. This has been taken advantage of from the practical standpoint by using citric acid in the form of eye drops or ointment in conjunction with the miotics to lower the p_H (raise the hydrogen ion concentration) of the fluids in the conjunctival cul-de-sac. There may also be a direct effect of the acid on the conjunctiva, as the reaction level of this tissue appears to be raised by such additions. It may be that other hydroxy acids are equally efficacious in this respect, but my clinical experience has been limited to only one, citric acid. I have had no experience with

the new antihistaminic substances, either prophylactically or therapeutically, in such reactions.

Eosinophilia in the conjunctival secretions does not occur in physostigmine or pilocarpine conjunctivitis but does occur in atropine and homatropine conjunctivitis, a point of difference the significance of which has been somewhat controversial. The opinion has been expressed in some quarters that, while atropine may produce an allergic reaction in the eyes, as evidenced by the occurrence of eosinophils, the absence of these cells in pilocarpine and physostigmine conjunctivitis would indicate that the reaction produced by the latter drugs is toxic rather than allergic. Preservatives used in commercial eye lotions have occasionally been observed as the cause of persistent ocular irritation, which disappears when the wash is discontinued. Chlorobutanol heads the list of offenders in my experience. Aqueous acriflavine solution, which is one of the most efficient and least irritating antiseptics for local use in the eye, occasionally produces a vascular reaction, with associated papillary swelling and the picture of an allergic reaction. One of the newer drugs which has found great favor as a conjunctival antiseptic is sodium sulfacetimide, which may, like other drugs, produce a local reaction in the eye. Many drugs have no adverse effect in the beginning but produce reactions after a time, as a cumulative effect or as a result of a change in the reactive capacity of the patient's tissues, in which they become less tolerant to the drug. This, in turn, is apparently influenced by the p_H of the tears.

Edema of the eyelids and urticarial lesions about the eyes may occur as an acute allergic phenomenon due to the ingestion of a food or drug to which the patient is specifically hypersensitive. Optic neuritis and retinal hemorrhages have been attributed to allergic reactions by Hansel [1] and Wittich [2] and are regarded as counterparts of urticarial lesions elsewhere on the body.

Stings from a variety of insects, such as mosquitoes and bees, are also productive of acute edematous lesions of the eyelids, owing in part to the chemical which they inject and also, at least in sensitized persons, to protein bodies contained in the poison. There are instances on record in which constitutional reaction associated with such stings have been fatal, probably as a result of allergic shock.

Acute or chronic vascular engorgement of the conjunctival vessels may be the result of exposure to an unsuspected atopic reagin and may be the cause of long-standing conjunctival hyperemia, or red eyes.

1. Hansel, F. K.: Allergy of the Nose and Paranasal Sinuses, St. Louis, C. V. Mosby Company, 1936.

2. Wittich, J. W.: Journal-Lancet **65**:249, 1945.

The precise disease picture produced in allergic reactions in the eye depends largely on which ocular tissue is acting as the shock organ.

Physical allergy is that form of allergic reaction produced in susceptible persons by exposure to heat, light, cold or trauma. Pure physical allergy is considered rare but as an exaggerating factor added to other allergic reactions is often of importance. The release of histamine-like substances in the skin is considered the most probable mechanism involved in the production of physical allergy. In the Southwest it is not uncommon to encounter patients with chronic conjunctival irritation which is apparently due solely to the effect of heat and bright sunlight. My personal belief is that this type of physical allergy is a prominent factor in pterygium, though almost certainly other, equally important, factors, such as a shift in the hydrogen ion concentration of the tears to the alkaline side, which operate in the production of this condition. Recurrence of pterygiums following adequate surgical removal is not infrequent in the Southwest, most probably because no environmental change is made in the physical surroundings after the growths are removed. A continuance of the underlying factors originally producing the hyperplastic change in the conjunctiva will explain the recurrence of pterygiums in most persons. The use of lenses which afford a degree of protection from infra-red rays, as well as from the ultraviolet and visible light, will afford a degree of protection against these physical factors and are to be considered as part of the postoperative care of patients with pterygium, even though no refractive error is present. A persistent highly alkaline reaction of the tears no doubt favors the return of pterygiums. The use of a mild acid type of eye lotion will, in my opinion, act as a deterrent or an inhibitor to the return of these growths. The objection to aqueous lotions is that their effect is fleeting, lasting one an average less than two minutes. Ointments of citric acid in aquaphor® (an oxycholesterol-petrolatum ointment base) mixed with petrolatum are more lasting, and on the whole more advisable, but from the patient's standpoint, less so because of the greasiness and blurred vision associated with their use.

"Spring catarrh," or vernal conjunctivitis, frequently encountered in children, is usually, if not always, associated with persistent exposure to atopens. It is characterized by chronic irritation with catarrhal discharge and follicular hypertrophy. The severity of the reaction in the eyes is governed by the degree of susceptibility in the patient, the quantity or dose of the atopen, the persistence of contact with it and its degree of solubility in the tears, the last factor, in turn, being influenced by the degree of alkalinity of the tears. It will be recalled that the Calmette reaction for tuberculosis, or the so-called ophthalmic

reaction, was one of the early reactions to tuberculin elicited in persons sensitized to tuberculin (tuberculotoxin), that test being performed by dropping a tuberculin solution into the eye. A more or less violent vascular response, produced in the eye of the sensitized person, constituted a positive diagnostic reaction. The test was a crude one, and not without danger to the eye. Fortunately, this test is no longer used on the human subject, as far safer and more exact diagnostic procedures have supplanted it.

In follicular or in vernal conjunctivitis, the discharge from the eye is heavy with eosinophils, a fact of importance in differential diagnosis of follicular conjunctivitis and other conditions likely to be confused with it, such as trachoma. In general, it may be said that the eosinophil is the key cell in the cytologic diagnosis of allergy of the eye, as it is in allergic rhinitis—just as edema may be said to be the characteristic lesion of allergy. Trained observers will not be satisfied with the occurrence of a few pink-stained leukocytes in the smear; to be diagnostic, the cells must contain visible eosinophilic granules.

Herbert[3] in 1903 noted the importance of the eosinophil in the diagnosis of vernal conjunctivitis but did not associate the finding with atopism or allergy. This came later. Basophilic leukocytes were also noted as being increased in allergic conjunctivitis and vernal catarrh by Thygeson,[4] who observed that they sometimes outnumbered eosinophils in the latter condition. He further stated that the presence of basophils in large numbers appeared to have as definite a diagnostic significance as that of eosinophils. Fragmentation of eosinophils and basophils is frequent in the discharge of allergic conjunctivitis; therefore, free eosinophilic or basophilic granules are often seen in the secretions. The intact cells are not always easily demonstrated, but persistence will almost always be rewarded with success. The cells in question are more easily demonstrated in conjunctival curettings or scraping than in the free secretions. Currettement is accomplished by anesthetizing the lids by instillation of one of the usual anesthetics and using a Myerhofer curet to obtain the surface cells. Excessive currettage is neither necessary nor advisable. The material for examination is spread on an ordinary glass slide and fixed by drying in the air. Polychrome stains, such as the Wright or the Giemsa stain, are commonly employed in staining such preparations. My colleagues and I have preferred to make a percentage count of the leukocytes when possible, as it gives an excellent check on the clinical course of an

3. Herbert, H.: Brit. M. J. **2**:735, 1903; **2**:1232, 1907.

4. Thygeson, P.: Cytology of Conjunctival Exudates, The Association for Research in Ophthalmology, 15th Annual Report, pp. 7, 1946.

allergic reaction and is a good index of the efficiency of any particular type of therapy. This is easily accomplished by inserting a cardboard diaphragm in the microscope eyepiece, in which a 0.5 cm. or, if preferred, a 1 cm. square window has been cut. Such percentage counts are made in nasal as well as in ophthalmic smears; 50 to 100 cells are counted if they are present in sufficient number, fragmented cells being disregarded. Often, the percentage in ophthalmic smears must be computed on a total count of as few as 25 cells. A decrease in percentage of eosinophils usually means clinical improvement.

"Generally speaking, polymorphonuclear neutrophils are characteristic of bacterial infections, mononuclear cells of virus infections, eosinophiles and basophiles of allergy." [5]

Phlyctenular conjunctivitis and keratoconjunctivitis are at present regarded by most authorities as an allergic response to bacterial protein from a focus of infection, such as might occur about the teeth or in the tonsils or the nasal accessory sinuses. Such attacks not infrequently follow acute infections of the upper respiratory tract, such as an ordinary cold or influenza, the theory being that the eye has previously been rendered hypersensitive to the bacterial toxin or virus in such infections and a second exposure to a toxin from the same or a closely related strain of bacteria produces the local explosion in the eye. No eosinophils occur in the conjunctival secretions in this disease. Bacterial toxins or proteins apparently do not have the property of calling forth an eosinophilic response in tissues sensitized to them. I have recently observed a severe bilateral phlyctenular keratoconjunctivitis in a 16 year old girl in which the corneal infiltrate predominated; this condition resisted all measures, including tonsillectomy, but responded promptly to hyposensitization with house dust and ragweed pollen.

A clear concept of what is meant by the Arthus and Shwartzman phenomena is essential to a proper understanding of what takes place in certain allergic reactions. Briefly stated, they may be defined as reactions occurring in a previously sensitized structure after the second administration or absorption of the original type of antigen which produced the initial sensitization. When a bacterial antigen acts as the sensitizing agent, the antigen producing the second reaction may come from either an identical or a closely related strain of bacteria.[5]

According to Carleton,[6] up to 1943, there were reported 46 cases of lenticular cataract occurring in persons with chronic neurodermatitis in which the relation was explainable on an allergic basis. The change in the lens usually comes in the fourth decade of life.

5. Gill, E. K.: Texas State J. Med. **43**:330-334 (Sept.) 1947.
6. Carleton, A.: Brit. J. Dermat. **55**:83, 1943.

Tuberculosis, so far as the ocular response is concerned, may be considered a disease in the allergic category. The nucleoprotein or tuberculin elaborated in the growth of the tubercle bacillus may act as a foreign protein to sensitize the eye, and later, with continued absorption, it acts as a secondary allergin to produce any one of a variety of inflammatory lesions in the eye. Eosinophilia is not produced in the secretions of the eye by the lesions of ocular tuberculosis. In tissue sections a lymphocytic response and giant cells are seen, but in the secretions of the tuberculous eye there is no diagnostic cytologic picture. The fact that the organism is sensitized to tuberculoprotein or to tubercle bacillus toxin can be demonstrated by any one of several well known tests—the patch test, the scarification test or the intradermal injection of some form of tuberculin. The most frequently used tuberculins are old tuberculin and the purified protein derivative of tuberculin. Extremely minute quantities of tuberculoprotein will precipitate reactions, local, general and focal, in sensitized persons. Great caution must be exercised in their use, as the employment of too large doses in the injection test may lead to severe and disastrous focal reactions in an eye which is sensitized to this substance. The tuberculin used in testing, regardless of its type, must be enormously diluted, for once it is introduced into the body it cannot be withdrawn nor can its effect be mitigated. No one should use tuberculin as a diagnostic agent unless thoroughly familiar with its potential dangers. The patch test is easily applied, more easily controlled and far safer in all respects, and is considered best for the occasional investigator, though it is perhaps a little less desirable from the diagnostic standpoint than the injection test, as it lacks the quantitative features of the latter.

In syphilis the reactions induced in the eye, such as iritis, iridocylitis and keratitis, are considered allergic manifestations, due to the infected person's becoming sensitized to his own strain of syphilitic toxin or antibodies—certainly to some protein-like substance elaborated in the course of the disease. The mechanism of production is similar to that of tuberculous lesions of the eye.

Elschnig,[7] and later Wood,[8] developed the theory that sympathetic ophthalmia was an allergic manifestation due to disintegration of uveal pigment and its absorption, with resultant autosensitization. Further absorption of uveal pigment precipitates a secondary reaction wherever the particular type of pigment (melanin) occurs. A sterile or allergic

7. Elschnig, A.: Arch. f. Ophth. **75**:459, 473, 1910; ibid. **76**:509-546, 1910; ibid. **78**:549-583, 1911.

8. Wood, A. C.; Arch. Ophth. **45**:557-563, 1916; ibid **47**:161, 1918; ibid. **46**:503-517, 1917; Immune Reactions Following Injuries to Uveal Tract, J. A. M. A. **77**:1317-1322 (Oct. 22) 1921.

reaction is produced in the uveal pigment layer of the opposing, or sympathizing, eye by this mechanism. Other observers supported the view of Elschnig and Wood and Knapp.[9] To any one who has seriously investigated the relation of uveal pigment to sympathetic ophthalmia the relationship is quite evident, and so striking that the logical conclusion seems to be that, while sympathetic uveitis may not be entirely an allergic disease, there is an allergic phase in which a definite sensitization to uveal pigment exists. This may be further substantiated by a complement fixation test of the patient's blood, in which uveal pigment is used as the antigen. This test does not lend itself to routine clinical use because of its complex nature, but it is interesting from the investigative standpoint. Local hypersensitiveness to injected uveal pigment and clinical response to pigment used therapeutically are additional substantiating evidence to support the belief that there is an allergic phase of this disease. Hypersensitiveness to uveal pigment is preceded by a phase in which bacterial toxins pave the way and prepare the eye for sensitization by producing foci of inflammation in the choroid. Such foci arise as a result of the eye's having become previously sensitized by bacteria or toxins from the same or a closely related strain of organisms elaborating the original sensitizing allergin or toxin. The reaction which accompanies such pathologic changes in the choroid of the exciting eye is sufficient to cause disintegration and solution of a certain amount of uveal pigment, which is absorbed and sensitizes the eyes—both of them—to uveal pigment. Subsequent absorption of pigment, even remote, precipitates a secondary reaction of an allergic nature, which is designated as sympathetic ophthalmia. The reaction in the first, or exciting, eye is more correctly designated sympathogenic ophthalmia or uveitis. In eyes enucleated prior to the actual development of sympathetic ophthalmia, characteristic microscopic changes exist, consisting of foci of lymphocytic infiltration with associated giant cell formation. Eosinophils have been found in the choroid in practically all my sections of eyes removed as the exciting eye in cases of sympathetic ophthalmia, but not in sufficient number to be unequivocally diagnostic of allergy.

Further substantiating evidence of the specificity of uveal pigment in sympathetic ophthalmia is obtained by histologic examination of the point of injection in the cutaneous test, with the demonstration of phagocytosis of pigment particles in the excised area of injection after a two week interval has elapsed, as noted by Friedenwald,[10] and,

9. Wood, A. C., and Knapp, A.: Arch. Ophth. **51**:560-565, 1922; Bull. Johns Hopkins Hosp. **33**:419-425, 1922. Knapp, A.: Tr. Am. Ophth. Soc. **22**: 256-268, 1924.

10. Friedenwald, J. S.: Am. J. Ophth. **17**:1008, 1934.

further, by study of the opsonic index with pigment, as recommended by Henton.[11]

In the type of uveitis of a nonsympathetic nature due to focal infection, we have concluded from our investigation with uveal pigment, both diagnostic and therapeutic, that in most cases this type of uveitis has essentially the same immunologic background as has sympathetic ophthalmia.[12]

The question arises as to why one person will respond with an allergic reaction of the eye from any cause and another, with apparently the same clinical background, will fail to exhibit such reaction. This is a question which must be answered largely speculatively, but certain factors are apparently of some importance in formulating an answer.

Heredity plays an important part in certain allergic disease of the eye. Follicular conjunctivitis is one of these. It is not at all uncommon to encounter several children in the same family who are suffering from conjunctival folliculosis, in the same or in varying degrees. Not infrequently one of the parents will give a history of similar trouble in childhood. It is easy in such instances to make the mistake of considering such ocular lesions as trachomatous and assuming that the children are all infected with the trachoma virus. Hay fever, or allergic rhinitis, is a frequent accompaniment of follicular conjunctivitis, revealing the patient's general allergic tendency. The spontaneous disappearance of conjunctival folliculosis at puberty is strongly suggestive of an influence of the sex glands, which take on certain changes at that period.

Alkalization of patients with certain allergic manifestations, such as hay fever, accentuates the symptoms; and it has been observed clinically that when a patient is hovering on the verge of an allergic explosion, such as allergic rhinitis, the reaction can usually be precipitated by the internal administration of alkalis. I have cause to believe that in such patients the body chemistry is too far on the basic, or alkaline, side, even though this shift may be very slight and represented by only a minute variation in the hydrogen ion concentration (p_H) of the tissues. It is further believed that the difference in the p_H (acid-base balance) inherent in different persons may explain why in one person a certain disease, such as conjunctival folliculosis or even sympathetic ophthalmia, will develop and in another it will not, even though all other factors are apparently operating equally. The use of an acid ash diet or the ingestion of diacid salts and even mineral

11. Henton, H. C., Opsonic Index for Uveal Pigment in Treated Patients, Arch. Ophth. **17**:113-116 (Jan.) 1937.

12. Gill, William D.: South M. J. **23**:885-894, 1930; ibid. **34**:959-968 (Sept.) 1941; Ann. Otol., Rhin. & Laryng. **44**:486-492 (June) 1935.

acids, such as hydrochloric acid, has a salutary influence on the course of most allergic states, while the employment of alkalis and alkaline ash foods has the opposite effect, an observation which may be advanced as further support of this belief.

Verhoeff and Lemoine [13] described a type of allergic reaction in the eye which they termed phacoanaphylactic endophthalmitis, in which the patient is sensitive to lens protein. The hypersensitiveness to lens protein is said to be present in 8 to 10 per cent of all persons. The reaction occurs when lens protein is allowed to remain in the eye after the lens capsuls is broken, as would occur in a combined linear cataract extraction, in needling operations to produce absorption of juvenile cataract or in certain types of accidental trauma to the eye. The reaction varies in degree, being slight in some instances and intense in others; often it is sufficiently severe to cause loss of the eye. Such a reaction may be anticipated by preoperative intradermal testing with lens protein (antigen). The condition is to be avoided by using the intracapsular method of lens extraction in all cases of cataracts in which this technic is applicable. Avoidance of such a reaction is one of the strongest arguments in favor of this method. If the combined linear method of extraction is used, the anterior chamber must be cleared as thoroughly as possible of all residual lens material at the time of operation, either by expression or by irrigation. In cases of juvenile cataract the rapid method of eliminating the lens diminishes the possibility of the occurrence of such a reaction, but does not with certainty prevent it. Failure of operation in many cases of juvenile cataract is traceable to failure to consider the potential possibilities of absorption of lens protein in such cases. Precautionary measures may forestall such reactions—preliminary skin tests and preoperative desensitization with lens protein if indicated by the test; if the reaction occurs after cataract extraction or a needling operation, active hyposensitization with lens antigen is advised, usually in conjunction with a secondary operation to remove every vestige of lenticular matter.

With the advent of the plastic artificial eye, we have noted that a surprising number of persons who were wearing such eyes experienced abundant discharge from the socket, associated with discomfort and intense swelling and edema of the conjunctiva. The reaction subsides when the plastic eye is removed and is allowed to remain out of the socket for several days, but returns when the plastic eye is reinstated. Substitution of an old-fashioned glass eye does not cause reappearance of the reaction, but each time the plastic eye is inserted the reaction recurs, and the socket reassumes its former irritated state in a very

13. Verhoeff, F. H., and Lemoine, A. N.: Endophthalmitis Phacoanaphylatica, Tr. Internat. Cong. Ophth., Washington 1922, pp. 234.

short time. This is cited as a form of atopism or allergy to the plastic material which ophthalmologists must be on guard to detect. Cytologic findings in the secretions in such cases have not been altogether consistent, nor have a sufficient number of cases been observed, and long enough, to justify a positive statement of the diagnostic value of the cytologic findings in the discharge in such reactions, but the indication is, most strongly, that certain of the plastics used in the manufacture of plastic eyes produce an allergic reaction, characterized by edema in the mucous membrane of the socket and a marked discharge. When such reactions occur, the offending prosthesis should be removed and the old-fashioned glass eye substituted, or another eye must be made from an entirely different plastic material, which, in turn, may or may not produce a reaction in the socket. Some patients may be sensitive to all types of plastic material but may be able to wear an old-fashioned glass eye with complete comfort and without any adverse reaction.

SUMMARY

Allergic reactions are of frequent occurrence in the eye and are of many kinds.

The etiologic factors are numerous.

Cytologic examination of the secretions from the eye is a definite aid in diagnosis in certain, but not in all, allergic conditions affecting the eye.

Attention is called to reactions in the eye caused by plastic prostheses, a condition which may be considered a form of atopism, or contact allergy.

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ANISOCORIA

Attempted Induction by Unilateral Illumination

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THE VALUE of anisocoria as a diagnostic sign has been limited by reports of normal or physiologic anisocoria in as high as 40 per cent of all patients.[1] Löwenstein's pupillographic method has eliminated a large number of cases of these so-called physiologic anisocorias through detailed cinematographic analysis under both static and dynamic conditions.[2] In order to realize the potentialities of this method, and in order to place the proper value on the finding of anisocoria, it is necessary to delimit sharply that which may be physiologic from that which is pathologic. Toward this end the present study has been undertaken.

Several theories of the course of the afferent pupillary fibers have been proposed in attempts to explain various phenomena of the pupillary movements.[3] The most generally accepted schema is that proposed by Behr,[3c] who stated the belief that the macular fibers from each retina have equal representation in the constrictor nuclei of both sides, but that most of the extramacular fibers from each side are represented in the constrictor nucleus of the same side. That is, a greater number of extramacular fibers come from the nasal portion of the retina, cross in the chiasm and also decussate centrally. Magoun and Ranson[4] have shown the existence of a partial decussation of the pupillary paths in the posterior commissure. Whether such a decussation results in an unequal representation of extramacular fibers in the constrictor nuclei, as hypothesized by Behr, still lacks anatomic proof.

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1. Walter: Ueber transitorische Pupillendifferenz bei Nervengesunden, Neurol. Zentralbl. **35**:619, 1916.

2. Lowenstein, O.: Pupillographic Studies: Present state of Pupillography; Its Method and Diagnostic Significance, Arch. Ophth. **27**:969 (May) 1942.

3. Bernheimer, S.: (*a*) Die Reflexbahn der Pupillarreaktion, Arch. f. Ophth. **47**:1, 1899. (*b*) Levinsohn, G.: Beiträge zur Physiologie des Puppillenreflexes, Arch. f. Ophth. **59**:436, 1904. (*c*) Behr: Die Lehre von den Pupillenbewegungen, Berlin, Julius Springer, 1924.

4. Magoun, H. W., and Ranson, S. W.: The Afferent Path of the Light Reflex. Arch. Ophth. **13**:862 (May) 1935.

Abelsdorff and Piper [5] reported anisocoria on unequal illumination of the eyes, the eye receiving the greater light having the smaller pupil. Their work was done with the illumination directed into the eyes along the visual axis in a manner similar to that used in the present study. Behr [3c] stated that the pupil of an eye blinded from a lesion of the retina or the optic nerve is larger than that of the seeing eye if the two halves of the retina of the seeing eye are equally illuminated. These two situations differ only in degree, since the blind eye may be likened to an eye in total darkness, with the fellow eye receiving light. From Behr's schema, the explanation of the phenomenon is as follows: The macular fibers have equal bilateral representation; most of the extramacular fibers of the brightly lighted eye cross in the chiasm, then cross again in the intercalated neuron, ending in the constrictor nucleus of the homolateral side; the remainder of the extramacular fibers of this eye do not cross in the chiasm but cross in the intercalated neuron to end in the constrictor nucleus of the opposite side; therefore, the constrictor nucleus of the homolateral side receives a stronger stimulus. Weiler, cited by Behr,[3c] was able to induce anisocoria as thus described in only 6 per cent of cases. However, the observations of Abelsdorff and Piper and others to the contrary, as well as Behr's explanation, have been carried over in more recent textbooks.[6] Only the most pertinent references are cited here. A fuller presentation of the literature has been given by Behr.[3c]

The present experiments attempt, under conditions of simulated unilateral blindness, to investigate the production of anisocoria as described in the preceding paragraph. Purely nasal or temporal illumination has been purposely avoided.

METHOD

Apparatus similar to that used by Löwenstein [2] was used in these experiments. A motion picture camera with double lenses recorded the pupils simultaneously side by side on 35 mm. film. The exposures were made at the rate of 10 per second. The film used was sensitive to infra-red rays and a basal infra-red illumination was maintained constantly on each eye. All experiments took place in a dark room.

The patients were adapted to darkness for fifteen minutes, after which pictures were taken to record the amount of anisocoria in darkness. Light reactions and phychosensory reflexes were performed and recorded to determine whether the responses for each patient fell within normal limits. The attempted induction of anisocoria was carried out as follows: A beam of blue-green light (Wratten filter XI), having a diameter of 4 cm. at the eye, was turned on the right eye

5. Abelsdorff, G., and Piper, H.: Vergleichende Messungen der Weite der direkt und der consensuell reagirenden Pupille, Arch. f. Augenh. **51**:366, 1905.

6. Spiegel, E. A., and Sommer, L.: Neurology of the Eye, Ear, Nose, and Throat, New York, Grune & Stratton, Inc., 1944.

from a distance of 33 cm. and at an angle 5 degrees above the visual axis; lateral displacement with regard to the visual axis was avoided; successive illuminations of 0.02, 12.0 and 92.0 foot candles were used; pictures were taken after six minutes' exposure to each of the first two intensities and after three minutes' exposure to the third intensity; the right eye was then returned to darkness, and the beam of the third (high) intensity was directed on the left eye for six minutes, and pictures were again taken.

PRESENT STUDY

Twenty-five normal adult patients were examined, 9 of whom were males and 16 females. The anisocoria exhibited in darkness by these patients is shown in table 1. The average difference in darkness was 0.21 mm.

TABLE 1.—*Amount of Anisocoria in Darkness (25 Cases)*

Anisocoria (mm.)	0.01-0.10	0.11-0.20	0.21-0.30	0.31-0.40	0.41-0.50	0.51-0.60	0.61-0.70
Number of patients	8	4	6	3	3	0	1

TABLE 2.—*Changes in Anisocoria After Unilateral Illumination of Various Intensities*

	No Change	More Anisocoria	Less Anisocoria	Stimulated Pupil Relatively Larger	Stimulated Pupil Relatively Smaller
A. Low Intensity (24 Cases)					
Number of patients	1	10	13	9	14
B. Medium Intensity (25 Cases)					
Number of patients	0	13	12	9	16
C. High Intensity (25 Cases)					
Number of patients	1	9	15	11	13

Table 2 A shows the changes found after six minutes' illumination of the right eye with dim light. The change in total anisocoria is less significant than the change of the stimulated pupil relative to its fellow. That is, a patient whose right pupil was larger in darkness might show decreased anisocoria after unilateral illumination of the right eye, indicating a relatively greater constriction of the stimulated pupil. On the other hand, if the left pupil had been larger in darkness, decreased anisocoria after illumination of the right eye would indicate a relatively greater constriction of the unstimulated pupil. In the 9 cases in which the stimulated pupil became relatively larger, the average increase was 0.15 mm. In the 14 cases in which the stimulated pupil became relatively smaller, the average decrease was 0.12 mm.

Table 2 B shows the changes found after six minutes' illumination of the right eye with light of medium intensity. There is little difference between these figures and those in part A of the table. In the 9 cases

in which the stimulated pupil became relatively larger, the average increase was 0.12 mm. In the 16 cases in which the stimulated pupil became relatively smaller, the average decrease was 0.12 mm. It should be noted that at the end of this series the stimulated eye had been under continuous illumination for twelve minutes.

Table 2 C shows the changes found after three minutes' illumination with the light of high intensity. At the conclusion of this series the eyes had been under light for fifteen minutes without interruption. In these results a lessened anisocoria in a significant number of patients is revealed. This may perhaps be explained as a function of the diminished size of the pupil under bright light. That is, an anisocoria which remains fairly constant in amount in the middle range of pupillary play decreases

TABLE 3.—*Changes in Anisocoria After High Intensity Illumination of the Opposite Eye (23 Cases)*

	No Change	More Anisocoria	Less Anisocoria	Stimulated Pupil Relatively Larger	Stimulated Pupil Relatively Smaller
Number of patients.....	0	8	15	9	14

TABLE 4.—*Analysis of Cases with Relatively Smaller Stimulated Pupil (57 Cases)*

Intensity	No. of Cases of Relatively Smaller Pupils	Percentage of Total	Amount of Relative Constriction, Mm.						
			0.01-0.10	0.11-0.20	0.21-0.30	0.31-0.40	0.41-0.50	0.51-0.60	0.61-0.90
Low (right).....	14	58	9	4	0	0	1	0	0
Medium (right).....	16	64	12	2	1	0	0	0	1
High (right).....	13	52	8	2	1	1	0	1	0
High (left).....	14	61	7	3	2	0	1	1	0

as the extremes of dilation or constriction are approached. Each iris may receive constrictor impulses in excess of the number needed for maximum constriction, so that the pupillary diameters may be very nearly the same, even though the imbalance of the constrictor impulses still exists. In the group of 11 relatively larger stimulated pupils, the average increase was 0.09 mm. In the 13 relatively smaller stimulated pupils, the average decrease was 0.15 mm.

Table 3 shows the changes after six minutes' illumination of the previously unlighted (left) eye with light of high intensity. It shows no essential differences from table 2 C and is susceptible of the same interpretation. In the 9 cases in which the stimulated pupil became relatively larger, the average increase was 0.14 mm. In the 14 cases in which the stimulated pupil became relatively smaller, the average decrease was 0.16 mm.

In table 4 the relative constriction of the stimulated pupils is analyzed for the whole series of cases. It shows that in 57 per cent of the cases the stimulated pupil constricted more, and more than half did so at every level of illumination. Of the 57 such instances, the relative constriction was less than 0.20 mm. in 47, or 82 per cent. This is less than the average anisocoria of 0.21 mm. exhibited in darkness by the whole series. If all the patients had shown no anisocoria in darkness, and if the same behavior on illumination had been shown, then a slight anisocoria, usually of less than 0.20 mm. would have developed in 57 per cent, with the lighted pupil smaller. Such was not the case. In this series an actual increase in anisocoria occurred in only 42 per cent, and in only 1 of these was the amount (0.88 mm., relative) grossly appreciable.

SUMMARY AND CONCLUSIONS

In a series of 25 cases attempts were made to induce anisocoria by unilateral illumination at three levels of intensity.

The average difference between the pupils in the dark-adapted patient was 0.21 mm. Only 1 subject showed an amount sufficient to be detected reliably by ordinary observation.

The change in the amount of anisocoria was approximately the same for all levels of illumination, namely, 0.02, 12 and 92 foot candles.

An actual increase in the amount of anisocoria was seen in 42 per cent of cases, while a relative increase, averaging 0.20 mm., was seen in 57 per cent. This apparently indicates a chance variation.

Contrary to the observations of Abelsdorff and Piper [5] and the statements of Behr,[3c] the illuminated pupil did not constrict more than the unilluminated pupil. These results are similar to those of Weiler.[3c]

It appears that uniform illumination of one eye within the limits of time and intensity of this study may be disregarded as a cause of anisocoria. Since the technic of the experiments closely approximated that used by Löwenstein,[2] it follows that a factor of induced anisocoria is not inherent in his method.

It should again be emphasized that these studies were confined to illumination of the central portion of the retina and did not include stimulation of the temporal or nasal part of the retina.

Dr. Otto Löwenstein gave help and guidance in this study.

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CEPHALOCELE OF POSTERIOR PART OF ORBIT

General Survey, with Report of a Case

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BY CEPHALOCELE of the posterior part of the orbit is understood cerebral hernia arising from the middle cerebral fossa and penetrating into the posterior part of the orbit behind the bulbus oculi, through a preformed or nonpreformed opening in the wall of the orbit.

Posterior cephalocele of the orbit belongs to the group of basal cephaloceles. Besides the basal type, by far the rarest, there are occipital, frontal, parietal and lateral cephaloceles. Cephalocele of the anterior portion of the orbit, generally arising from the nasofrontal region, belongs to the frontal cephaloceles and is much more frequently observed than is the posterior lesion. Stadfeldt,[1] who was the first in Denmark to assess the available material, found in the literature up to 1902, 20 cases of anterior and 8 cases of posterior cephalocele. It has been the subject of some discussion whether a certain case (Oettinger's [2]) of the latter type should be counted as belonging to that group or be reckoned among the anterior cephaloceles. Schmidt,[3] who in 1927 reported a case, besides surveying available reports, did not include his tumor among the posterior cephaloceles. Nor is this case included in the present report, because, as first stated by Peters,[4] it appears from the description that the deficiency of the orbital wall was located so anteriorly that the lesion may reasonably be supposed to have originated from the nasofrontal area, and not, as asserted by Stadfeldt,[1] from the fossa media cranii.

Reali collected 140 cases of cephalocele, with the following distribution: occipital, 86 cases; frontal, 33 cases; parietal, 12 cases; lateral, 8 cases, and basal, 1 case. Estimates by Houel, Lawrence, Wallmann,

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From the Eye Department of the University Hospital (the Rigshospital); Chief, Prof. H. Ehlers, M.D.; and the Neurosurgical Department of the University Hospital; Chief, Prof. E. Bush, M.D.

1. Stadfeldt, A.: Ueber die Cephalocele der Augenhöhle, Nord. med. Arkiv., 1903, vol. 3, pt. 1, no. 12; vol. 4, no. 20.

2. Oettinger: Klin. Monatsbl. f. Augenh. 1874.

3. Schmidt, M.: On Multiple Cephaloceles in the Fossa Cranii Media, Acta psychiat. et neurol. **3**:265, 1927.

4. Peters: Ueber einem Fall von doppeltseitiger Encephalocele der Orbita, Klin. Monatsbl. f. Augenh. **59**:553, 1917.

and Larger and Lindfors are in agreement with Reali's distribution of favorite sites, whereas Deloff found the number of frontal cephaloceles to exceed the number of occipital cephaloceles.

Some dispute has arisen as to the term cephalocele, since the hernial content has not always consisted of cerebral tissue in cases with characteristic symptoms and in cases in which deformities of the bone, demonstrated by postmortem or by roentgenographic examination, were of the nature of hernial openings. Other designations have therefore been suggested and applied for short periods, the aim being a specification of the hernial content—meningocele, encephalocele, meningocephalocele, hydroencephalomeningocele and encephalocystomeningocele. The last designation was proposed by Muscatello, who stated the opinion that the condition was caused by cystic arachnitis, a view which he, among others, supported on the basis of microscopic changes in biopsy tissue of the cephalocele (epithelial proliferation, cystic degeneration and invasion of vessels).

In the present survey, comprising 31 cases of cephalocele of the posterior part of the orbit, only cases have been included in which the symptomatology was characteristic and in which roentgenologic or postmortem examination of the cranium established the presence of a hernial formation containing substance of the middle cerebral fossa and extending into the orbit behind the bulbus, either through a defect of the orbital wall or through an enlarged, preformed opening, as well as cases in which explorative operation of the orbit revealed tumors consisting of brain tissue—with or without membranes and with or without cystic degeneration—and cerebrospinal fluid.

In view of the comparatively high number of congenital cephaloceles and the frequency of unilateral deformities of bone in the form of incomplete ossification and the fact that in a single case only did meningeal symptoms precede the diagnosis of symptoms of cephalocele, it appears to me more reasonable to consider the cystic degenerative changes in the arachnoid (*a*) as secondary to the actual defect through which the hernial formation penetrates the orbit, and (*b*) as a natural consequence of the altered pressure and gradual compression which is associated with the hernial opening, similar to that which may be seen during operation for incipient incarcerated hernia. Nor does the fact that, of 8 patients who died after operation, degenerative, inflammatory changes of the meninges were observed in 7 support the view that this involvement of the meninges is the fundamental, primary disturbance. On the contrary, from the reports, it appears that in these 7 patients the meningeal symptoms appeared after operation, presumably as a consequence of exogenous infection (the cases in question are from a series of 14 operative cases, 5 of incisions, 7 of excisions and 2 of punctures).

The preformed openings through which cephalocele of the posterior portion of the orbit may penetrate are the optic foramen, the sphenoidal and sphenomaxillary fissures and the posterior ethmoidal foramen. They may be either of normal size or enlarged. In general, enlargements are unilateral, and they may affect all openings or one only.

The nonpreformed openings may be (*a*) congenital deficiencies (incomplete ossification, generally unilateral, or birth trauma), (*b*) traumatic defects of later origin or (*c*) defects caused by preexisting or concurrent disease. Listed in the order of their frequency, they are: erosion of bone due to aneurysms, malignant tumors, inflammatory processes, angiomas, generalized osteitis fibrosa cystica, osteitis deformans an internal hydrocephalus.

As a rule, the soft meninges and brain tissue form component parts of the hernial formation, whereas the dura mater is usually not involved in the later stages of the disorder but, bursting early in the development of the hernia, forms, together with the edges of the bone, part of the hernial opening. Only in the early stages, in which the lesion is not generally well developed yet is marked by symptoms characteristic of cephalocele of the posterior part of the orbit, does the dura mater form part of the hernial sac.

As a further sign that in by far the greater proportion of cases the primary disturbance is an abnormal development in early fetal life, the apparent existence of a gradual transition may be emphasized. It would appear likely that this transition starts in the manner exemplified in Borochovic's [4a] cases (1930): One of his patients, the fourth of 4 siblings, a girl aged 7, had double anophthalmos with orbital cyst, and her sister (sibling 3) had anophthalmos on the right side and microphthalmos on the left side, both patients having pronounced congenital defects of the roof, as well as of the posterior wall of the orbit. The transition might pass through the stage illustrated in the case described by Tauber in 1900 (with unilateral microphthalmos and incomplete ossification of the posterior wall, the roof and the floor of the orbit, so that the cranial cavity, the orbit and the maxillary sinus of the affected side formed a single cavity) to that in the case reported by Cohen,[5] with microphthalmos and enlargement of the optic foramen on the same side. It may further be emphasized that the microphthalmos which appears in 4 of the 31 cases collected here must be considered the result of abnormal development, like the cranial malformations, rather than as a secondary change consequent to the increased pressure in the orbit. Further, the disease is characterized, in most instances, by

4a. Borochovic, S.: Kongeniter Anophthalmus mit Orbitalcyste, Ukrain. ophth. Ztschr. **3**:122, 1930.

5. Cohen, M.: Orbital Meningo-Encephalocele Associated with Microphthalmia, J. A. M. A. **89**:746 (Sept. 3) 1927.

being a congenital disorder, which may become manifest or aggravated as the result of trauma or the presence of an intercurrent disease. The condition progresses slowly. In isolated cases only does it manifest itself by serious symptoms, and rarely by general cerebral symptoms, such symptoms, when present, being epileptic fits, defective speech and impaired memory.

The material on which the present survey is based comprised 36 cases, reported as instances of cephalocele of the posterior orbit. From these must be eliminated 5 cases: (1) a case reported by Oettinger,[5] in which the possibility that the cephalocele originated in the anterior cranial fossa cannot with certainty be excluded, as, in addition to a deficiency corresponding to the ethmoid bone, the orbital plane was to all intents and purposes absent; (2) a case reported by Schousboe[6]; (3) a case reported by Kalt[7]; (4) a case reported by Tietze[8] before a German meeting of ophthalmologists in Munich in 1935, and (5) a case reported by Bonnet[9] in 1937. In the same year, however, Aranowich protested against the classification of Bonnet's[9] case with the cephaloceles, since it was clearly one of hernia of the dura mater. With respect to cases 2, 3 and 4, it may be said that the information available is too scanty to permit with certainty their inclusion in the group of cephaloceles of the posterior orbit. The remaining 31 cases were collected from the available literature published between 1841 and 1948. The cases concern 15 females and 9 males; for 7 patients there was no information as to sex. The ages of the patients at first examination ranged from 13 weeks to 77 years. By far the greater proportion were young persons: Twenty-two patients were under 30 and 3 over 30, while there is no statement as to the age of 6 patients. In 21 cases in which the patients were especially interrogated, there was no familial disposition to ocular diseases or congenital malformations. In 20 cases the condition had been accompanied with symptoms since shortly after birth, though to a comparatively slight degree. In 2 cases there had been serious trauma (Lücke and Tauber), pulsating exophthalmos quickly developing after a blow to the right eye in 1 case and blindness following head trauma, sustained at the age of 15 in the other. In the latter progressive symptoms of cephalocele of the posterior orbit had been present since birth, whereas in the former the patient had always been

6. Schousboe, F.: Méningo-encéphalocèle de l'angle supérointerne de l'orbite; excision; mort, Bull. Soc. d'opht. de Paris 1927, pp. 162-164.

7. Kalt, E.: Un cas d'encéphalocèle orbitaire, Bull. Soc. d'opht. de Paris, 1930, pp. 220-266.

8. Tietze, H.: Ein Fall von Meningoencephalocele der Orbita mit rudimentärem Augenanlage: Report, Gesellschaft für süddeutsch Augenärtze, München, 1935.

9. Bonnet, F.: Meningoencephalocele der Augenhöhle, Semana méd. **1**:1025, 1937.

healthy up to the time of the accident and free from ocular disease. In 13 of 31 cases the initial examination revealed the tumor of the orbit, most frequently located medial to and passing behind the bulbus oculi. In all cases the tumors were soft, yielding and slightly elastic. In 20 cases the bulbus was displaced in relation to its original position in the orbit, being displaced downward and laterally in 18 cases and downward and medially (Wheeler [10] and Jaensch [11]) in only 2 cases. In Jaensch's [11] case there was, in addition to the anomalous posterior wall of the orbit, a defect in the lateral wall, through which the cephalocele had made its way. In 15 cases the mobility of the bulbus was limited.

In 28 cases there was unilateral exophthalmos, the condition being fairly equally distributed between the right and the left eye. In 26 cases exophthalmos was of pulsating type, the beats being of varied intensity and synchronous with the carotid pulse in all cases.

In 13 cases it was possible to correct the protrusion of the bulbus by slight pressure on the eyeball, resulting in simultaneous further protrusion of other contents of the orbit but without producing cerebral symptoms. In 3 cases attempts at reposition of the globe caused vertigo or nausea (Gala [12]; Schmidt and Jensen [13]; Strandberg). In no case did changes in the pulsation of the bulbus appear with the variation in the position of the head when the subject was in an upright position, while in 1 case Jaensch [11] reported considerable reduction in pulsation of the bulb after a prolonged period of quiet rest on the back. In 3 cases only was it possible to decrease the pulsation of the bulbus by compressing the carotid artery.

Kubli [14] mentioned aggravation of the pulsating exophthalmos on coughing, sneezing and forced contraction of the abdominal muscles, observations which correspond well with those of Gala to the effect that protrusion decreases in proportion to the quantity of cerebrospinal fluid withdrawn by lumbar puncture and that the cerebrospinal pressure increases when reposition of the protruding bulbus is attempted. Wheeler [10] observed that the pulsation of the eyeball disappeared and the protrusion diminished in the tribromoethanol anesthesia.

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10. Wheeler, J. M.: Pulsation of the Eyeball Associated with Defects in the Wall of the Orbit, Bull. Neurol. Inst., New York **5**:476, 1936.

11. Jaensch, P. A.: (*a*) Encephalocele orbitae posterior, Klin. Monatsbl. f. Augenh. **76**:433, 1926; (*b*) Encephalocele orbitae posterior, Med. Klin. **1**:450, 1928; (*c*) Hydrocephalus congenitus und Cephalocele orbitae posterior, Zentralbl. f. d. ges. Ophth. **20**:208, 1928; (*d*) Cephalocele orbitae posterior, Klin. Monatsbl. f. Augenh. **107**:561, 1941.

12. Gala, A.: Encephalocele bei M. Recklinghausen, Zentralbl. f. d. ges. Neurol. u. Psychiat. **28**:64, 1922.

13. Schmidt, L., and Jensen, F.: Un cas d'encéphalocèle postérieure d'orbite, Arch. d'opht. **39**:108, 1922.

14. Kubli: Encephalocele unterm obern Lide, read before the Petersburg Ophthalmological Association, April 8, 1910.

Palpebral edema was present in 16 cases—the palpebra superior being involved in 13 cases and the palpebra inferior in 3 cases. Ptosis existed in 8 cases.

In 18 cases the size of the eyeballs was stated to have been normal, whereas in 4 cases microphthalmos (Taube, Delpeuch,[15] Parson and Coates[16] and Cohen[5]) was present. Furthermore, in Cohen's case reduced tension and horizontal nystagmus were reported. In 7 cases the affected eyes had reduction of vision from 6/12 to total blindness. In 3 cases (Dandy,[17] Jaensch[11] and Wheeler[10]) diplopia was present, and examination showed changed visual fields in 2 cases (Dandy,[17] slight concentric contraction; Jaensch,[11] annular scotoma).

Ophthalmoscopic examination revealed changes in 5 cases. In 3 cases (Jaensch,[11] Dandy[17] and Stadfeldt[1]) there were overfilling and tortuosity of veins. In 1 case (Lagleyse[18]) papillary edges were blurred, and in 1 case (Parson and Coates[16]) there was coloboma of the optic nerve.

Among other ocular symptoms were interstitial keratitis, in 1 of Jaensch's cases; atrophy of the iris, cataract and stiff pupil, in 1 of Cohen's, and coloboma of the iris, in 1 of Stadfeldt's. In 2 cases there was audible vascular bruit, objectively demonstrated in 1 and subjectively felt in the other.

Cranial changes were many and characteristic: Enlargement of the orbit on the affected side, in 21 cases; visible asymmetry of the cranium, in 12 cases, and prominence of the temporal region, in 9 cases. In 4 of these 9 cases, in addition to posterior cephalocele of the orbit, there was cephalocele of the temporal region (Jaensch,[11a, d] Schreyer and Sprenger[19] and Strandberg). Two of these cases deserve further mention. In the case published by Jaensch[11a] there was incipient cephalocele auricularis, examination at the ear clinic revealing a tense, pulsating prolapse of the dura mater in the external auditory canal. Schreyer and Sprenger's patient also had cephalocele auricularis, but the hernial opening of the cephalocele in the temporal region was not formed by suture edges or by deficiencies corresponding to a suture, as is usually seen, but by the temporal squama directly, in the middle of which the lesion was located.

In 7 cases there was enlargement of the optic foramen, and in 6 cases enlargement of the sphenoidal fissure, whereas the posterior

15. Delpeuch, P. E.: Traité des tumeurs de l'orbite, Arch. d'opht., 1860.

16. Parson and Coates: A Case of Orbital Encephalocele with Unique Malformations of the Brain and Eye, Brain **39**:108, 1906.

17. Dandy, W. E.: An Operative Treatment for Certain Cases of Meningocele (or Encephalocele) into the Orbit, Arch. Ophth. **2**:123 (Aug.) 1929.

18. Lagleyse: Arch. d'opht., 1900, cited by Stadfeldt.[1]

19. Schreyer, W., and Sprenger, W.: Ueber basale Cephalocelen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:252, 1927.

ethmoidal foramen was enlarged and formed the hernial opening in only 1 case (Zitowskij [20]). In 12 cases there were palpable deficiencies in the orbital wall.

Roentgenographic or postmortem examinations in 15 cases revealed deficiencies in the orbital wall; these were located in the posterior wall in 8 cases, in the most posterior part of the roof in 4 cases, in the side wall in 3 cases (in the medial wall in 2 cases and in the lateral wall in 1 case (Jaensch [11]) In 2 cases the sella turcica was enlarged. Other defects causing cephalocele of the posterior part of the orbit were the absence, in 5 cases, of the lesser wing and/or parts of the greater wing of the sphenoid bone (Jaensch [11] [1928 and 1941], Ecklentz,[21] Scullica [22] and Strandberg). In 2 cases there appeared, simultaneously, a cephalocele from the posterior cerebral fossa. In both cases the defect was located in the occiput (Jaensch [11] and van der Hoeve [23]). In 6 cases there were reported fistulas from the ventricular system to the cephalocele. The most serious defect was described in Tauber's case, with imperfect ossification of the lesser wing of the sphenoid bone, of the base of the greater wing of the sphenoid bone, of the cribriform plate of the ethmoid bone and of the upper and lower orbital walls.

The most frequent ocular symptom of cephalocele of the posterior part of the orbit is a slowly developing, unilateral exophthalmos, which appears chiefly in younger persons and, in the majority of cases, pulsates synchronously with the pulse and is reducible by pressure on the bulbus, usually without accompanying cerebral symptoms but sometimes associated with vertigo and nausea. The exophthalmos may change in relation to the cerebrospinal pressure, whereas the pulsation in the majority of cases remains unaffected by compression of the carotid artery on the affected side. The eyeball is displaced, most frequently downward and laterally, but in few cases downward and medially if the deficiency is located in the lateral wall of the orbit. Movement of the eyeball is hampered, the degree being dependent on the size of the encephalocele. There is a palpable, soft, elastic tumor in the orbit. There is edema of the eyelids, most frequently of the upper and in some cases of the lower lid. Ptosis, reduced vision and microphthalmos must in all likelihood be regarded as due to defective development, in the same manner as are the skeletal deficiencies, rather than as secondary

20. Zitowskij, M.: Zur Kasuistik der orbitalen cerebralen Hernien, Sovet. vestnik. oftal. **7**:249, 1935.

21. Ecklentz: Klin. Monatsbl. f. Augenh., 1901; cited by Stadfeldt.[1]

22. Scullica, F.: Esoftalmo da meningocele della fossa cranica media, Ann. di ottal. e clin. ocul. **2**:734, 1927.

23. van der Hoeve, J.: Schädeldefekte bei Hydrocephalus, Ophthalmologica Valencia **1**:237, 1935.

phenomena; further, there may be diplopia, central changes in the form of venous congestion, incipient papilledema and coloboma of the optic nerve, when the hernial opening of the cephalocele is formed by an enlarged optic foramen. In isolated cases there are changed visual field, bruit, reduced tension (observed only in cases with concurrent microphthalmos) and nystagmus.

OPERATIVE TREATMENT

Operative treatment was reported in 23 cases. In 3 cases ligation of the carotid artery on the affected side was done, with no improvement. In 5 cases incision of the palpable orbital tumor was carried out. In 7 cases incision of part of the hernial formation was performed (in 1 case, without ligation of the hernial sac). In 2 cases the sac was punctured. Of the cases, 14 in all, in which incision, excision or puncture was carried out, death was reported to have followed operation in 8. In 7 cases the cause was postoperative meningitis, and in 1 case, "cerebral crisis." In 4 cases transplantation was carried out with living tissue. In 1 case trepanation was done, with dural substitute, and in 1, craniotomy, with use of gel film. In the last-mentioned 6 cases the patients survived, and the symptoms were noted to improve; the bone healed well in the 4 cases of transplantation.

Although the number of operations carried out is small and the descriptions in several cases are incomplete, the results indicate that patients are best served by operations similar to those for hernial lesions; i. e., craniotomy, reposition of the contents of the hernia and substitution according to the defect—bone transplantation in smaller defects; gel film, of a thickness suitable to the size of the defect, in larger ones, or dural substitute, if and when practicable.

REPORT OF CASE

A case of cephalocele of the posterior portion of the orbit from the University Hospital, Copenhagen, is reported.

A girl aged 6 had a condition diagnosed as pulsating exophthalmos and was referred to the ophthalmologic department of the hospital on Feb. 23, 1948.

She had had the usual diseases of childhood without complications. There was no familial disposition to ocular disease or congenital malformations. She was the last of 4 siblings. She had previously been in good health, and her mental and physical development corresponded to the normal for her age.

The present disorder had been observed by her mother during the last year; it manifested itself by pulsation of the right eye in the orbit, synchronous with the pulse, as well as increasing asymmetry of the cranium, with thickening of the right temporal region. No other abnormalities were observed by her mother.

Physical Examination.—The child was apparently normal and healthy. Physical and mental development was normal for her age. Stethoscopic examination showed a normal condition of the heart and lungs. The abdomen was soft and normal; there were no neurologic abnormalities.

Cranium: There was slight asymmetry of the face. The right temporal region showed a watch glass–shaped prominence. Corresponding to and along the sphenoparietal suture was felt a cranial defect, 3 to 5 mm. broad and just under 2 cm. long. No hernial formation was felt there, but pressure elicited a soft, elastic resistant mass, and the area was only slightly tender.

Ocular Examination.—Vision was 3/4.5 in each eye, without correction.

The right orbit was larger and more ovoid than the left. No palpable deficiency of the orbital wall was noted. There was slight protrusion of the right eyeball, which pulsated synchronously with the pulse, with varying magnitude, the maximum pulse being 3 mm. and the minimum scarcely visible. Neither the protrusion nor the pulsation was altered by rotation of the head. During repeated attempts to compress the carotid artery of the affected side, it appeared once as though the pulsation of the eyeball was decreasing; it returned, however, before the compression was discontinued.

Fig. 1.—A girl aged 6 with cephalocele of the posterior part of the right orbit. *A* shows symmetry of the face, with prominence of the right temporal region and the right eyeball in a more lateral and downward position than the left. *B*, patient seen from the side at an oblique angle, showing prominence of the lower frontal part of the right temporal region.

Exophthalmometric measurements (Hertel) were 15 mm. on the right side and 13 mm. on the left side. The protrusion could be reduced by pressure, thereby causing slight prolapse of the content of the upper portion of the orbit, accompanied with a slight feeling of discomfort, slight nausea and uncharacteristic vertigo.

No bruit was audible on auscultation of the cranium. There was no nystagmus or double vision. The right bulbus could be moved freely in all directions, to the same extent as the left eye. The right eyeball was displaced slightly downward and laterally. There was no strabismus. The cornea, conjunctiva, anterior chamber, lens and iris were normal in each eye.

Ophthalmoscopic examination revealed a normal condition on both sides.

Roentgenographic Examination (Dr. Gilg).—The cranium was normal in shape and size. The sella turcica was normal. The left orbit was normal, whereas the right orbit was a little larger and more ovoid; corresponding to the

entire background of the orbit was a large defect, the upper edge of which was clearly discernible, whereas its lower limit was difficult to locate. The defect seemed to affect great proportions of the wings of the sphenoid bone. No signs of defect appeared elsewhere in the cranium. The paranasal sinuses were weakly developed.

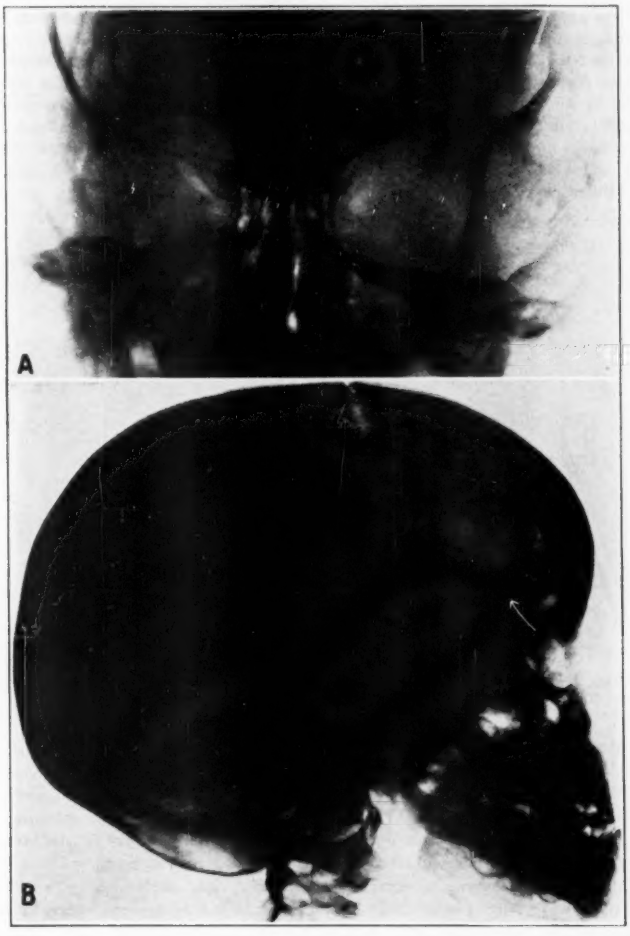

Fig. 2.—*A*, roentgenogram of the cranium in the frontal plane. The right orbit is larger and more ovoid than the left. There is no sphenoidal fissure on the right side or visible posterior wall of the right orbit. *B*, roentgenogram taken in the right lateral position. The right sphenoidal wing is strongly dislocated upward and lies like an arc over the right orbit.

Roentgenographic examination (Prof. F. Møller) of the cranium in the frontal plane repeated nine days later showed that the right orbit was larger and more ovoid than the left. Here it was not possible to see the posterior wall or the sphenoidal fissure, and the wings of the sphenoid bone appeared to be displaced upward to a considerable degree, lying like an arc over the upper part of the orbit. The same phenomenon was noted on special exposure of the optic foramen, which in itself presented no abnormalities. A roentgenogram in the right lateral position revealed the same displacement, and one received the impression that pressure was being exerted by an unknown factor, which had produced the dislocation and caused erosion of the posterior lateral wall of the orbit. No abnormalities of the paranasal sinuses could be demonstrated, but the frontal ethmoidal sinuses were very incompletely developed.

Planigrams of the right orbit were then attempted, but the pictures were not so valuable as they might have been because the patient moved during exposures.

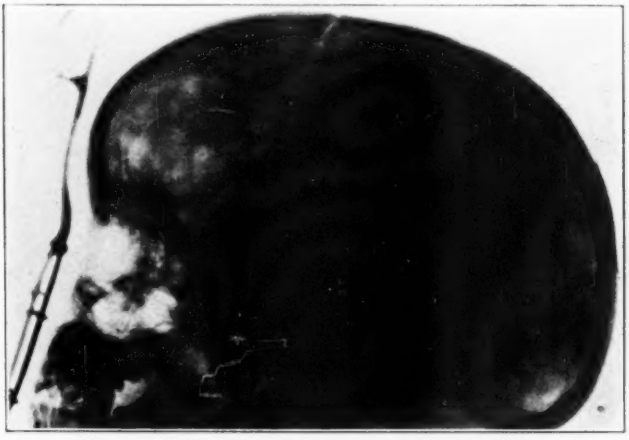

Fig. 3.—Arteriogram of the right side, showing compression of the carotid sinus on the right side. There is no sign of aneurysm.

March 19, 1948: The patient was moved to the neurosurgical department of the University Hospital for arteriography and possible operation. Here, ophthalmologic and neurologic examinations gave the results previously noted.

March 22: Arteriograms (Prof. Busch) revealed a peculiar prominence of the carotid siphon on the right side, which may have been produced by the examination, but the presence of partial carotid block was beyond doubt.

March 23: A ventriculographic examination was performed. The fluid pressure was 140 systolic and 100 diastolic. Roentgenograms showed no abnormalities. Operation was decided on and was performed by Prof. E. Busch.

Operation.—A frontal cutaneous incision was made over the anterior part of the right hemisphere. The tumor of the temporal region appeared to be only a blister in the bone over a strongly protruding temporal lobe. The wing of

the sphenoid bone was in an extreme frontal location, only 2 cm. from the orbital process, and followed the middle meningeal artery, which was about 2 mm. thick. The fissure of Sylvius was very deep, whereas the bone itself, especially the wing of the sphenoid bone, was as thin as paper. It was removed laterally by forceps and the dura mater was opened. It was then possible to penetrate to the middle fossa and to the posterior wall of the orbit, where it appeared that no bone had formed; only a thin, incomplete membrane of connective tissue separated the middle fossa, corresponding to the temporal pole, from the orbital contents. Exploration was carried through the frontal lobe to the internal carotid artery, which was peculiarly twisted in relation to the dura mater, but there were no signs of tumor or aneurysm. The posterior wall of the orbit was covered with a gel film, and the dura was completely sutured. The wound was closed after the piece of bone on the temporal side had been slightly reduced.

Postoperative Course.—The patient stood the operation well. The postoperative course was uncomplicated.

On March 30 the patient was moved back to the ophthalmologic department; she felt entirely well. A faint pulsating exophthalmos was still present, though to a considerably less pronounced degree. Dislocation of the eyeball was decreasing. The patient was discharged on April 16, to report at intervals for continued control.

SUMMARY

On the basis of 31 cases of cephalocele of the posterior part of the orbit collected from the literature between 1841 and 1948, the most prominent pathologicoanatomic changes are reviewed. Various reasons are cited in support of the concept that this lesion is a congenital malformation, caused by failure in ossification, rather than a condition following cystic degenerative arachnitis, a view previously held. Directions for treatment are given. In conclusion, a recently diagnosed Danish case is reported.

Prof. H. Ehlers, M.D., drew my attention to this case and encouraged me in the writing of the present report.

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GONIOTOMY IN TREATMENT OF CONGENITAL GLAUCOMA

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THE RESULTS obained from goniotomy in the treatment of 16 eyes (9 patients) with congenital glaucoma are reported. The outcome in these eyes is comparable to the reported by Barkan.[1] All the patients discussed in this paper have been followed for one and one-half to three years after operation.

HISTORICAL REVIEW

The treatment of congenital glaucoma has been discouraging in the past. Favorable response to miotics is rare. The multiplicity of operations in use in the various clinics speaks for their inadequacy. Anderson,[2] in his monograph, pointed out that congenital glaucoma is one of the chief causes of blindness in children. He cited surveys from several countries which showed that 2.4 to 13.5 per cent of children admitted to schools for the blind had lost their eyesight because of congenital glaucoma. He concluded his monograph by dwelling on the poor outlook for patients with congenital glaucoma and stated that there is little hope of preserving vision sufficient for earning a livelihood.

The excellent results from goniotomy reported by Barkan afforded a striking contrast. Incision of the angle of the anterior chamber for the treatment of congenital glaucoma was first reported by de Vincentiis in 1893.[3] Anderson[4] stated that several Italian and French ophthalmic surgeons have since used the operation in the treatment of congenital glaucoma. Some good results were apparently obtained, but the operation has never become popular or widely used.

Read at the Eighty-Fifth Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., June 2, 1949.

From the Department of Ophthalmology, Hospital of the University of Pennsylvania and University of Pennsylvania School of Medicine.

1. Barkan, Otto: (*a*) Operation for Congenital Glaucoma, Am. J. Ophth. **25**:552-568 (May) 1942; (*b*) Technic of Goniotomy for Congenital Glaucoma, Tr. Am. Acad. Ophth. **52**:210-226 (Jan.-Feb.) 1948.

2. Anderson, J. R.: Hydrophthalmia, or Congenital Glaucoma, London, Cambridge University Press, 1939, pp. 4 and 5.

3. de Vincentiis, C.: Incisione dell'angolo irideo nel glaucoma, Ann. di ottal. **22**:540, 1893.

4. Anderson,[2] p. 312.

Barkan used the procedure on 17 eyes and first reported his results in 1942.[1a] The tension was normalized in 16 of the 17 eyes, and visual function was maintained in 14. In 2 of the 3 blind eyes, vision had been lost before surgical treatment was instituted. In only 1 eye, therefore, did operation fail to normalize the tension in time to save vision. In 1947,[1b] Barkan added the results obtained in 76 eyes with congenital glaucoma. In 66 of these the tension was normalized and vision maintained. All of the children had been followed from one to ten years. He concluded that the operation if performed early gave excellent results. He, therefore, made a plea for early diagnosis and prompt surgical intervention.

The results obtained in the treatment of congenital glaucoma at the Hospital of the University of Pennsylvania over a period of years had been highly disheartening. Because of these discouraging results and in view of Barkan's reports, my colleagues and I decided to try goniotomy, using it first in June 1946 on 2 eyes nearly blind from the disease (cases 1 and 2). The tension was not controlled, but since the operation seemed relatively safe we used it in more normal eyes.

TECHNIC

Operation was done with the patient under ether anesthesia. A Barkan knife[6] was used without aid of the goniotomy lens. We attempted to use the lens in the operation on 2 eyes, but it was cumbersome and contact with the cornea was difficult to maintain. The goniotomy knife was swept along from one-third to one-half the circumference of the eye, which was as much as could be reached at each operation. Miotics were used after operation in some eyes but seemed to have no effect on the outcome. Postoperative gonioscopy demonstrated scattered peripheral anterior synechias along the incised portion of the angle of a few eyes. More recently, therefore, we have filled the anterior chamber with air after goniotomy, employing the method outlined by Chandler.[5] An oblique incision is made with a knife needle in the lower temporal portion of the cornea through which air can be introduced with a fine needle, the tip of which not only enters the tract but goes through into the anterior chamber. All of our patients were reexamined at intervals of six weeks to three months during the first year after operation and then at least every six months. All tensions were recorded with the patient under general anesthesia.

PRESENTATION OF CASES

Case 1.—H. W. B., a 21 month old white boy, was admitted to the ophthalmologic clinic of the Hospital of the University of Pennsylvania on June 6, 1946. When he was 6 months of age, his pediatrician had first noticed that his eyes appeared abnormally large and referred him to an ophthalmologist for care. From that time until he was 21 months of age, a fifteen month period, seven paracenteses were performed on his right eye, the last in March 1946, and three on the left eye, the last in February 1946. The parents stated the belief that

5. Chandler, P. A.: Symposium Primary Glaucoma: V. Complications of Surgery, Tr. Am. Acad. Ophth. **53**:224-231 (Jan.-Feb.) 1949.

vision was poor in his right eye but that he could see well with his left eye. There was no family history of similar illness. He was a full term child. His mother had had no illness during her pregnancy. The child had always been healthy, and his development had been normal except for his eyes.

General physical examination revealed nothing remarkable. No other congenital anomalies were found. The reaction to a Kahn test was negative.

Examination of Eyes.—Visual Acuity: The child followed objects with the left eye only.

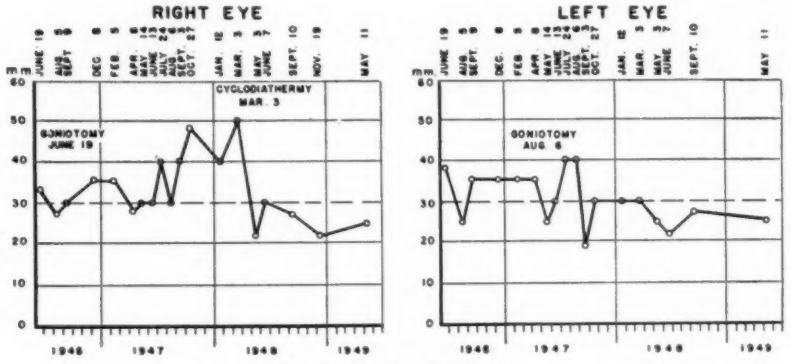

Fig. 1.—Tensions in case 1.

Fig. 2 (case 1).—Photograph taken at 4½ years of age, three years after goniotomy on the right eye, one year after cyclodiathermy on the right eye and approximately two and one-half years after goniotomy on the left eye.

External Examination: The lids were normal. Both palpebral fissures were somewhat widened. The corneas were definitely enlarged, the right more so than the left. Both corneas appeared clear to the naked eye. The anterior chambers were deep. The eyes were white. The right pupil reacted sluggishly to direct light; the left, normally.

Ophthalmoscopic Examination: Right eye: The media were clear except for lines in Descemet's membrane. The optic nerve was pale and deeply cupped. The retinal vessels were normal. The macula was healthy.

Left eye: This eye was similar to the right eye except that the optic nerve head was normal.

Ocular Tension: Tension was 33 mm. of mercury in the right eye and 38 mm. in the left eye (with a 7.5 Gm. weight and with the Schiøtz tonometer).

Gonioscopic Examination: Translucent embryonic tissue in the angle of the anterior chamber apparently displacing the iris toward the wall of the angle could be seen in each eye.

Impression: The impression was bilateral congenital glaucoma. Defective vision in the right eye was due to optic nerve atrophy.

Subsequent Clinical Course.—The child was admitted to the Hospital of the University of Pennsylvania for treatment. Because of Barkan's remarkable results with goniotomy, we elected to carry out this procedure on the right eye. Having had no experience with the procedure, we chose to do it on this eye, which was the patient's poorer one, having only questionable vision. Goniotomy was therefore performed, with the use of ether anesthesia, on June 19, 1946, without mishap. The eye showed little reaction. The child was discharged from the hospital two days later, to be observed subsequently in the outpatient department. The next ocular tension recorded was on Aug. 5, 1946, when it was 27 mm. of mercury in the eye operated on and 25 mm. in the left eye. Measurements of tension were made at regular intervals of about two months during the next year (fig. 1). The ocular tension in the surgically treated eye varied during that time from 27 to 40 mm. of mercury. The tension in the left eye was somewhat lower until July and August 1947, when it was recorded at 40 mm. of mercury on two successive occasions. The cornea was increasingly hazy.

By this time we had obtained apparently excellent results from goniotomy in several other patients with congenital glaucoma. We therefore recommended this procedure for his left eye, and the operation was performed on Aug. 6, 1947. Slight bleeding into the anterior chamber occurred, but the hemorrhage absorbed promptly and convalescence was uneventful. The tension of the left eye has since remained normal. The tension in the right eye, however, gradually rose until in March 1948 it was 50 mm. of mercury. In an attempt to save the eye, a cyclodiathermy was performed on March 3; since this time the tension has been stabilized at 22 to 30 mm. of mercury. Both corneas are at present clear (fig. 2).

Results of Goniotomy.—In the right eye, goniotomy failed, probably due to the advanced state of the disease, and cyclodiathermy subsequently normalized the ocular tension. In the left eye, tension was controlled by goniotomy.

Case 2.—A. O. K., a 12 year old white boy, was first admitted to the ophthalmologic clinic of the Hospital of the University of Pennsylvania on May 20, 1946, because of poor vision in his left eye. This had first been observed four months previously during a routine examination of the eyes by the school physician. He also had had some discomfort in the eye. His right eye had always seemed normal. No history of previous ocular difficulty could be obtained. He had had measles at 5 years and mumps at 7 years of age. No family history of ocular disease could be elicited. The results of general physical examination were not remarkable.

Examination of Eyes.—Visual Acuity: Vision was 6/9 in the right eye and was limited to hand movements in the left eye.

External Examination: The eyelids were normal. The left palpebral fissure was slightly wider than the right, and the left eyeball was somewhat more prominent than the right. Both corneas were slightly large, the left, measuring 14 mm., being larger than the right (13 mm.). To the naked eye they appeared clear. Ocular rotations were full. The conjunctiva and sclera appeared healthy. The pupils were round and regular, but the left (5 mm.) was larger than the right (4 mm.). Both reacted promptly in convergence. The right pupil reacted promptly to direct light but poorly to indirect light. The left pupil reacted in the opposite manner, a response characteristic of damage to the left optic nerve.

Examination with Slit Lamp: The corneas were clear except for ruptures of Descemet's membrane in each eye.

Ophthalmoscopic Examination: Right eye. The disk was normal in color and outline. The physiologic cup was normal in size and depth. The retinal vessels were normal. The macula was healthy. No lesions could be seen.

Left eye. The findings were similar to those in the right eye except that the disk was very pale. A large glaucomatous cup was present, extending to the margin of the disk. The vessels dropped sharply over the edge. Arterial pulsation was seen at the edge of the cup.

Ocular Tension: Tension was 22 mm. (5.5 Gm. weight) in the right eye and 35 mm. in the left eye (7.5 Gm. weight).

Visual Fields: Right eye. The peripheral field was full to a 1/330 white test object, and the central field was full to a 1/1,000 white test object. The blindspot was normal. No scotoma could be elicited.

Left eye. Only a residual temporal field to light remained.

Impression: The impression was that of (1) congenital glaucoma, with spontaneous cure in the right eye, and congenital glaucoma, with optic nerve atrophy in the left eye.

Subsequent Clinical Course.—Pilocarpine nitrate, 1 per cent, and physostigmine salicylate, 0.5 per cent, were prescribed four times daily for the left eye, but this medication failed to reduce the tension, which continued to be between 36 and 42 mm. of mercury during the following week. DFP (di-isopropyl fluorophosphate), 0.1 per cent, once daily likewise failed, although extreme miosis resulted.

The child was therefore admitted to the hospital, where a goniotomy was performed on his left eye on June 21. A considerable hyphema occurred, which absorbed promptly. Tension at the time of his discharge from the hospital was 22 mm. of mercury in the eye operated on. The tension of the eye was never over 22 mm. of mercury.

After his discharge from the hospital, the tension in the left eye remained normal for only a short time. On August 5, it was found to be 35 mm. of mercury and subsequently has never been below 40 mm., and has been as high as 65 mm. of mercury. The goniotomy was judged a complete failure. The tension in the right eye has fluctuated between 17 and 22 mm. of mercury, apparently representing spontaneous recovery from congenital glaucoma.

Results of Goniotomy.—Goniotomy produced spontaneous cure in the right eye and failed to relieve the glaucoma in the left eye, probably owing to the advanced state of the disease.

CASE 3.—T. B., a 9½ month old white boy, was admitted to the ophthalmologic clinic, Hospital of the University of Pennsylvania, on March 19, 1946. His eyes had been large since birth, and he had always been extremely sensitive to light.

He was seen by an ophthalmologist when only a few days old and pilocarpine was prescribed. His eyes continued to become larger, however, and the sensitivity to light increased. His parents believed that his vision was very defective.

His delivery had been normal at full term. He had had no illnesses. His development was normal. His mother had been healthy throughout her pregnancy. There was no family history of ocular disease. The results of general physical examination were not remarkable. The reaction to a Kahn test was negative.

Examination of Eyes.—Visual Acuity: The patient followed a light with each eye.

External Examination: The eyelids were normal. Both corneas were greatly enlarged, measuring 14 mm. in diameter. The anterior chambers were deep. Each cornea was diffusely cloudy. Iridodenesis was seen in each eye. The pupils reacted to light. The conjunctivas were slightly injected. The scleras had a somewhat bluish appearance.

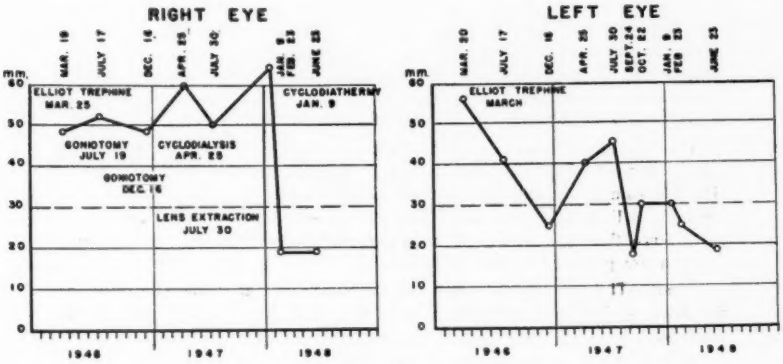

Fig. 3.—Tensions in case 3.

Examination with Slit Lamp: Ruptures of Descemet's membrane were present in each eye.

Gonioscopic Examination: The angles could not be seen because of a corneal haze.

Ophthalmoscopic Examination: A view of the fundus was also prevented by corneal opacity.

Ocular Tension: Tension was 48 mm. of mercury in the right eye and 56 mm. in the left eye (7.5 Gm. weight).

Impression: The diagnosis was bilateral congenital glaucoma.

Subsequent Clinical Course.—He was admitted to the Hospital of the University of Pennsylvania on March 19 for further treatment. At that time we were not yet doing goniotomies; so a bilateral Elliot trephination was done. Operation was performed on the right eye March 25 and on the left eye on March 29. The procedure was uneventful in the right eye, although the limbal tissue was found to be extremely thin. However, in the left eye vitreous presented in the trephine opening after the iridectomy. Persistent iridocyclitis developed in this eye,

and the cornea became opaque. The patient was discharged from the hospital two weeks later. A filtering bleb failed to develop in either eye. The tension was only temporarily lowered. He was subsequently followed in the outpatient department where the tension was found to rise gradually, until in July tension in the right eye was 52 mm. of mercury (7.5 Gm. weight) and that in the left, 40 mm. (fig. 3). The cornea of the left eye had become permanently opaque. He was readmitted to the hospital on July 19, 1946, because of the elevated tension, and a goniotomy was done on the right eye. A rather severe hyphema complicated the operation, but this absorbed during the next three weeks. He was again followed in the outpatient department, but the tension again gradually rose until it was 48 mm. of mercury on Dec. 16, 1946. Another goniotomy was done on the right eye, which was again accompanied with hyphema, which absorbed slowly. The tension was again lowered, but for only a short time, after which it again rose, this time to 60 mm. of mercury. We felt that his eye was probably too degenerated to be treated by goniotomy and, in desperation, resorted to cyclodialysis, on April 25. Severe hemorrhage in the anterior chamber ensued. The eye was then so badly damaged by the glaucoma and previous surgical procedures that the hemorrhage persisted two months. When absorption had taken place, it was observed that the lens in this eye was subluxated. Because the tension continued at 50 to 60 mm. of mercury, the lens was removed, with the thought that it might be contributing to the elevated tension. Extraction was accomplished successfully, without loss of vitreous, on July 30, 1947.

This procedure likewise failed to control the tension of the right eye, which was 64 mm. of mercury on Jan. 9, 1948. Because the child was having considerable pain and the parents wanted everything possible done to retain the eye and the light perception, which was still present, cyclodiathermy was carried out. Since then, the tension in his right eye has been within normal limits. The left eye gradually became phthisical.

Result.—The left eye was blind and phthisical, after the Elliot trephination; the right eye had light perception; goniotomy failed as secondary procedure in this eye.

Case 4.—N. S., a four month old boy, was seen through the courtesy of Dr. Francis Heed Adler, on March 28, 1946. The child had been photophobic from birth. His parents had noticed that his corneas had become cloudy during the preceding month. He had been born at full term, after an uneventful pregnancy. His development had been normal. There was no history of blindness in his family. The results of general examination were not remarkable. The reaction to a Kahn test was negative.

Examination of Eyes.—Visual Acuity: The infant was attracted by moving objects before either eye.

External Examination: The eyelids were normal. The palpebral fissures were slightly widened. Ocular motility was normal. Each cornea was enlarged, the right being about 13 mm. and the left 14 mm. in diameter. Photophobia was severe. The pupillary reactions to light were intact. The conjunctivas were hyperemic, but there was no discharge.

Examination with Slit Lamp: Both corneas were hazy. Ruptures of Descemet's membrane were present in each eye.

Ophthalmoscopic Examination: This examination could not be made because of the clouding of the corneas.

Ocular Tension: Tension was 38 mm. of mercury in the right eye and 35 mm. of mercury in the left eye (7.5 Gm. weight).

Impression: The diagnosis was bilateral congenital glaucoma.

Subsequent Clinical Course.—Bilateral Elliot corneoscleral trephination was done during the two weeks subsequent to his admission. At that time we had not as yet employed goniotomy in the treatment of congenital glaucoma. No operative complications were encountered, and convalescence was uneventful. Miotics were employed after operation, and the patient was reexamined at frequent intervals. Filtering blebs did not develop, and the tension of each eye was only temporarily controlled. On May 20 it was found to be 40 mm. of mercury in each eye (fig. 4). Both corneas were again cloudy and edematous. During June and July the cloudiness of the cornea continued and the tension as determined by palpation remained elevated in each eye.

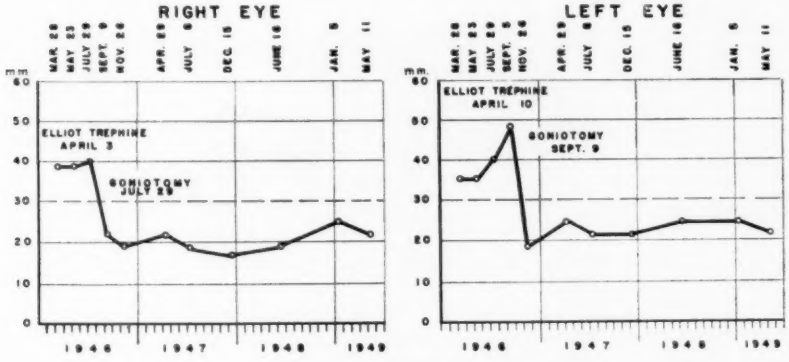

Fig. 4.—Tensions in case 4.

On July 29, therefore, a goniotomy was done on the right eye. Operation and convalescence were uneventful. Pronounced improvement in the eye resulted. The cornea of the treated eye cleared, but the intact (left) eye remained cloudy during the following six weeks. Because of the seemingly excellent result in the right eye, a goniotomy was performed on the left eye on September 9. The procedure was again carried out without complication. The child was seen at three month intervals after this operation until December 1947. Since then the intervals have been increased to six months. During this time the tension has been recorded as between 17 and 30 mm. of mercury. The corneas have remained clear.

After the goniotomies, ophthalmoscopic examination was easily carried out. The fundi were found to be very myopic, being seen with a —11.00 lens on each side. The disks were large. No glaucomatous cup or atrophy was present.

Postoperative gonioscopic examination revealed peripheral anterior synechias at the site of the goniotomy in the right eye. None were seen in the left eye. The child seems to have normal visual function except for high myopia (fig. 5).

Result.—Tension was controlled by bilateral goniotomy after corneoscleral trephination had failed.

CASE 5.—J. M., a 4 month old boy, was admitted to the ophthalmologic clinic of the Hospital of the University of Pennsylvania on Nov. 19, 1946. At birth his eyes were observed to be larger than normal, and he was sensitive to light. One week before he was brought to this clinic each cornea had become cloudy. His birth had been spontaneous, following a full term, uncomplicated pregnancy. Postnatal development had been normal. There was no history of ocular trouble or of blindness in the family. The general physical examinations revealed nothing remarkable. The reaction to a Kahn test was negative.

Examination of Eyes.—External Examination: The eyelids were normal. Ocular motility seemed full. Both corneas were enlarged, each being 13 mm. in diameter and definitely cloudy. The pupils reacted to light. The conjunctiva and sclera were healthy.

Gonioscopic Examination: The corneas were too cloudy to permit visualization of the angles.

Fig. 5 (case 4).—Photograph taken at 3½ years of age, two years and eight months after goniotomy on the right eye and two years and six months after goniotomy on the left eye.

Ophthalmoscopic Examination: Although fundus detail was difficult to see because of corneal haziness, the right disk appeared normal. The left nerve head seemed suspiciously cupped. The color of each disk was good.

Ocular Tension: Tension was 40 mm. of mercury in the right eye and 35 mm. of mercury in the left eye (7.5 Gm. weight).

Impression: The impression was that of bilateral congenital glaucoma.

Subsequent Clinical Course.—The patient was admitted to the Hospital of the University Pennsylvania, where a bilateral goniotomy was done on November 20. A small hyphema occurred in the right eye but none in the left eye. A small iridodialysis was produced at 2:30 o'clock in the right eye and at 7 o'clock in the left eye. One week later the tension was 12 mm. of mercury in the right eye and 25 mm. of mercury in the left eye. The corneas were almost clear. On December 9 the ocular tension was found to have risen to 35 mm. of mercury in the left eye; so another goniotomy was performed on this eye (fig. 6). On Jan. 3, 1946 the tension was 19 mm. of mercury in the right eye and 25 mm. of mercury in the left eye. The photophobia had disappeared. Typical ruptures were seen in Descemet's membrane of each eye. The child has been seen at regular intervals, and the tension has remained normal. His visual acuity seems excellent (fig. 7).

Result.—In the right eye tension was controlled by one goniotomy, and in the left eye, by two goniotomies.

CASE 6.—S. L., a 4 month old white girl, was first examined in the department of ophtalmology of the Hospital of the University of Pennsylvania on Jan. 24, 1947, at the request of the department of pediatrics.

She had been born with a diffuse port wine nevus involving both sides of her face and neck and the upper part of the thorax. Her eyeballs had been larger than normal at birth. Delivery had been spontaneous and the preg-

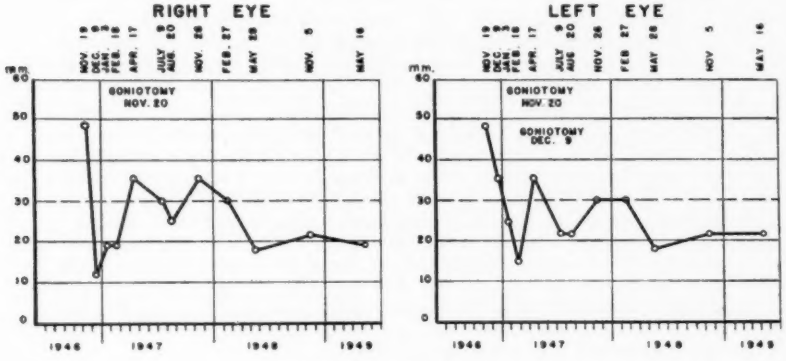

Fig. 6.—Tensions in case 5.

Fig. 7 (case 5).—Photograph taken at the age of 3 years, approximately two and one-half years after goniotomy.

nancy uncomplicated. The general physical examination showed nothing abnormal except for the port wine nevus. The reaction to a Kahn test was negative.

Examination of Eyes.—External Examination: The eyelids were normal except for a bilateral port wine nevus, part of the facial involvement. The palpebral fissures were slightly widened. The corneas were larger than normal, the right measuring 12.5 mm. and the left 13 mm. in diameter. The anterior chambers were deep. The pupils were equal and reacted promptly to light. Ocular motility seemed normal.

Gonioscopic Examination: A large number of anomalous blood vessels were noted in the angle of the anterior chamber of each eye, many of which extended on to the iris.

Ophthalmoscopic Examination: The fundi appeared normal except for suspiciously large physiologic cups. The color of the disks was normal.

Ocular Tension: Tension was 21 mm. of mercury in the right eye and 19 mm. in the left eye (7.5 Gm. weight).

Impression: A diagnosis of bilateral Sturge-Weber syndrome was considered, but the ocular tension did not confirm it.

Subsequent Clinical Course.—The parents were asked to bring the child in for another examination, which was done on March 29, 1947. At that time the corneas were hazy. The tension was 48 mm. of mercury in the right eye and 54 mm. in the left eye.

On March 31, bilateral goniotomy was performed. A rather severe hyphema occurred in each eye, the blood being absorbed during the week after operation. On April 7 the ocular tension was normal, but by April 27, although the corneas were clear, the ocular tension was again elevated. With the gonioscope anterior peripheral synechias were seen at the site of the goniotomy. Because of the presence of anomalous vessels in the angle, it was felt that the probable reason for the child's elevation in tension was anomalous vascularity of the uveal tract, related to the capillary hemangiomas of the face. Further goniotomy was therefore not carried out, but the child was referred to the department of roentgenology for irradiation of the eyes with a Philips tube. This was done and the child has been followed by us, in collaboration with the departments of roentgenology and pediatrics, since that time.

Within a short time after the beginning of roentgen therapy, the ocular tension fell to normal and has remained so since. The child is mentally retarded. In September 1948 she began to have convulsions, for which she was studied by the pediatrics department. It was thought that the seizures were due to an intracranial hemangioma, but roentgenograms of the skull, including an arteriogram, showed normal condition.

Result.—Goniotomy failed to control tension in either eye.

Case 7.—R. D., an 8½ month old boy, was first seen on May 26, 1947, through the courtesy of Dr. Samuel Phillips, Allentown, Pa. His parents had taken him to Dr. Phillips because they had noticed that the right eye was larger than the left and that he had seemed sensitive to light. Dr. Phillips made a diagnosis of buphthalmos. The child's history was otherwise not remarkable. The child was an identical twin, born spontaneously at full term. His twin brother was thought to have normal eyes. The results of general physical examination were noncontributory. The reaction to a Kahn test was negative.

Examination of Eyes.—External Examination: The eyelids were normal. The right cornea was larger than the left, the respective measurements being 11 and 10 mm. Both corneas were somewhat cloudy. The right anterior chamber was slightly deeper than the left. His pupils reacted normally. Ocular motility seemed normal. The conjunctiva and sclera were healthy.

Examination with the Slit Lamp: The right cornea was hazy. Epithelial bedewing was present. Tears were noted in Descemet's membrane.

Ophthalmoscopic Examination: No definite abnormality was found. The anatomic landmarks were difficult to evaluate.

Ocular Tension: Tension was 65 mm. of mercury in the right eye and 40 mm. in the left eye (7.5 Gm. weight).

Impression: The impression was that of bilateral congenital glaucoma.

Subsequent Clinical Course.—A bilateral goniotomy was done on May 26, 1947, and the operation was followed by a rather marked hyphema in each eye. The hemorrhages became absorbed in the next few days. The patient had been followed at intervals of three months or less since that time, and his ocular tension has been found to fluctuate between 17 and 30 mm. of mercury in each eye (fig. 8). His corneas have remained clear, and he uses his eyes normally (fig. 9).

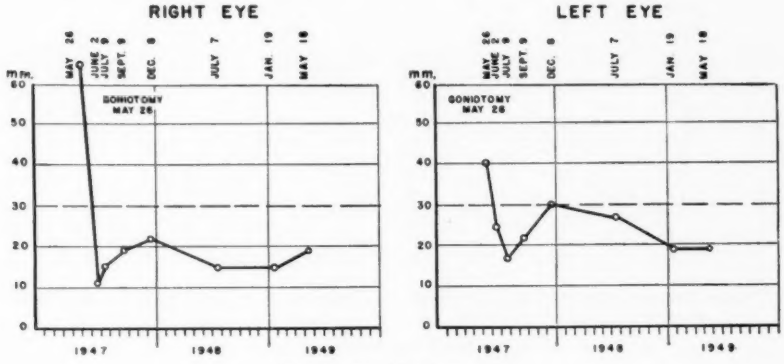

Fig. 8.—Tensions in case 7.

Fig. 9 (case 7).—Photographs taken at the age of 2½ years, two years after goniotomy.

Result.—Tension was controlled by bilateral goniotomy.

CASE 8.—R. D., an 8½ month old boy, was first examined on June 2, 1947, through the courtesy of Dr. Samuel Phillips, Allentown, Pa. The patient was referred for routine examination because his twin brother (case 7) had congenital glaucoma. The patient had no symptoms of ocular disease. Like his brother, he was born spontaneously at term. His development had been entirely normal.

The results of general physical examination were not remarkable. The reaction to a Kahn test was negative.

Examination of Eyes.—Visual Acuity: The patient used his eyes normally.

External Examination: No ocular abnormality was found.

Examination with Slit Lamp: The cornea, iris and lens of both eyes were normal. The aqueous was clear.

Ophthalmoscopic Examination: No definite abnormality could be found.

Ocular Tension: Tension was 40 mm. of mercury in the right eye and 40 mm. in the left eye.

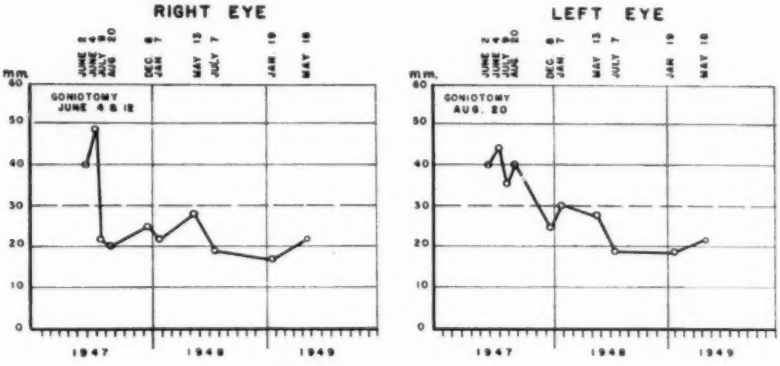

Fig. 10.—Tensions in case 8.

Fig. 11 (case 8).—Photograph taken at the age of 2½ years, nearly two years after goniotomy.

Impression: The impression was that of probable bilateral congenital glaucoma.

Subsequent Clinical Course.—The following day the tension was found to be 44 mm. of mercury in the right eye and 48 mm. in the left eye (7.5 Gm. weight). A goniotomy was therefore carried out on the right eye, which had the more elevated tension. Difficulty was encountered because of hemorrhage into the anterior chamber just as the incision was started. Operation was therefore deferred for eight days, until the eye had become quiet. The goniotomy was then completed in the right eye without difficulty. Because the patient ran a fever after operation, he was discharged from the hospital without having the

operation on the left eye. He was readmitted to the hospital in August. Tension was found to be normal in the right eye and 40 mm of mercury in the left eye (fig. 10). A goniotomy was therefore done on the left eye, without difficulty, on August 12. Convalescence was uneventful. He was discharged from the hospital in two days.

The patient also has been followed at regular intervals of no longer than three months. Tension has remained between 17 and 30 mm. of mercury, and his eyes are apparently normal (fig. 11).

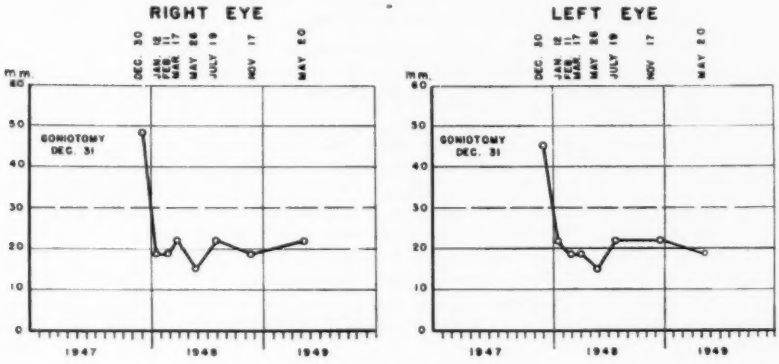

Fig. 12.—Tensions in case 9.

Fig. 13 (case 9).—Photograph taken at the age of 2 years, one and one-half years after goniotomy.

Result.—Tension was controlled by bilateral goniotomy.

Case 9.—W. B., a 5 month old boy, was seen on Dec. 30, 1947, through the courtesy of Dr. Samuel Phillips, Allentown, Pa. His eyes had been noticeably large since birth. The child had been extremely photophobic the month before admission and his corneas had become gray. The child was a fraternal twin. His sister's eyes were normal. There was no family history of ocular disease except for strabismus. The child was born at full term after a normal pregnancy.

Delivery was accomplished by low forceps. His development had been normal. The results of general physical examination were not significant. The reaction to the Kahn test was negative.

Examination of Eyes.—Visual Acuity: His eyes would follow a dim light.

External Examination: The palpebral fissures were slightly widened. Ocular rotations were full. Each cornea was large, measuring 13 mm. in diameter. The corneas were rather diffusely hazy. The anterior chambers were deep. The pupils reacted to light. The conjunctivas and scleras were healthy.

Examination with Slit Lamp: Bedewing of the corneal epithelium was present in each eye. There was some haziness in the stroma.

Ophthalmoscopic and Gonioscopic Examinations: These studies could not be made because of the corneal haze.

Ocular Tension: Tension was 45 mm. of mercury in the right eye and 48 mm. in the left eye.

Impression: The impression was that of bilateral congenital glaucoma.

Subsequent Clinical Course.—On Dec. 31, 1947, bilateral goniotomy was performed, with the patient under ether anesthesia. Convalescence was uneventful. The child was discharged from the hospital on January 3. Since this time he has been followed at intervals of six weeks to three months. His ocular tension has never been above 22 mm. of mercury in either eye (fig. 12).

Subsequent opthalmoscopic examination revealed that each disk was somewhat pale temporally and suspiciously cupped. Both corneas were clear (fig. 13).

Result.—Tension was controlled by bilateral goniotomy.

COMMENT

The results obained with goniotomy in the treatment of 16 eyes with congenital glaucoma (table) support the claims made for the procedure by Barkan. The tension was normalized in 11 eyes, and presumably good visual acuity was retained. The disease was so advanced in the 3 eyes in which goniotomy was unsuccessful that failure was not surprising. Two of the eyes were practically blind at the time of operation, and the third eye not only was greatly enlarged but had had an unsuccessful Elliot trephination prior to goniotomy. Goniotomy also failed to control the tension in either eye of a child who had bilateral congenital glaucoma associated with bilateral port wine nevus of the face and eyelids. This condition may represent a type of glaucoma which should not be treated by goniotomy, for it is generally agreed that the cause of this entity is a nevus-like involvement of the uveal tract. Preoperative gonioscopic examination of these 2 eyes demonstrated anomalous vessels over the ciliary body which extended into the iris; the presence of these vessels probably explained the rather severe hemorrhage caused by the goniotomy.

Goniotomy was done as a primary operative procedure in 8 of the 11 eyes in which it was successful. All the children were under 1 year of age at the time of operation. Ruptures of Descemet's membrane were absent in most of these eyes, although some degree of corneal

enlargement had occurred and corneal clouding was present in the eyes of all but 1 of the patients. The exception was a boy whose identical twin had bilateral congenital glaucoma with enlargement of one cornea. As a precautionary measure, his twin was examined. On two occasions the ocular tension was 40 mm. of mercury or above; a goniotomy, therefore, was carried out on each eye, although the corneas had not been clouded.

*Results of Goniotomy in 16 Eyes with Congenital Glaucoma**

Case No.	Eye	Age; Date of Operation	Pre-operative Tension, Mm. Hg.	Corneal Diameter, Mm.	Previous Operations	Result
1	OD	21 mo. June 19, 1946	33	14.0 (nearly blind)	Seven paracenteses	—
	OS	Aug. 6, 1947	38	13.0	Three paracenteses	+
2	OS	12 yr. June 21, 1946	35-40	14.0 (nearly blind)	None	—
3	OD	13½ mo. July 19, 1946	52	14.0 (nearly blind)	Trephination	—
4	OD	8 mo. July 29, 1946	40	13.0	Trephination	+
	OS	Sept. 9, 1946	40	14.0	Trephination	+
5	OD	4 mo. Nov. 20, 1946	40	13.0	None	+
	OS	Nov. 20, 1946 Dec. 9, 1946	35	13.0	None	+
6	OD	6 mo. Mar. 31, 1947	48	12.5 (Sturge-Weber Syndrome)	None	—
	OS	Mar. 31, 1947	54	13.0 (Sturge-Weber Syndrome)	None	—
7	OD	8½ mo. May 26, 1947	65	11.0	None	+
	OS	May 26, 1947	40	10.0	None	+
8	OD	8¼ mo. June 4, 1947 June 12, 1947	44	10.0	None	+
	OS	Aug. 12, 1947	48	10.0	None	+
9	OD	5 mo. Dec. 31, 1947	45	13.0	None	+
	OS	Dec. 31, 1947	48	13.0	None	+

* The results of goniotomy were successful (+) in 11 eyes and the operation failed (—) in 5 eyes, 3 of which were nearly blind and 2 had the Sturge-Weber Syndrome.

Claims for the successful treatment of congenital glaucoma, particularly when a small number of cases are involved, must necessarily take into consideration the occasional spontaneous resolution of the disease which does occur. An example was the right eye of A. O. K. (case 2), who at the age of 12 had normal tension in his right eye, vision of 6/9, a healthy optic nerve and a full visual field, in spite of definite corneal enlargement and classic ruptures of Descemet's membrane due to congenital glaucoma, which had cleared spontaneously. The fellow eye showed similar corneal enlargement with ruptures of Descemet's membrane but was nearly blind and had greatly increased tension with atrophy of the optic nerve.

Goniotomy possesses many advantages over other operations for congenital glaucoma. It seems very effective if done early. A second operation was done only once in our series. There seems to be little danger from the procedure. As pointed out by Barkan, the chief danger is hemorrhage into the anterior chamber. The blood is usually absorbed promptly. There is danger of injury to the ciliary body and of producing iridodialysis. No cases of sympathetic ophthalmia have been reported. Subluxation of the lens must be considered a possibility, particularly in enlarged eyes in which the zonular fibers are taut. In several of our patients peripheral anterior synechias developed after operation, but these adhesions can be avoided by the postoperative injection of air into the anterior chamber. We attempted to use the goniotomy lens twice in this series of cases but found that it offered no advantages and served only to make the operation more difficult.

The operation seems to be permanently successful. Patients reported on in this paper have all been followed one and one-half years or longer, and in none of the eyes in which the goniotomy was successful has the tension again become elevated. At the time of his last report, Barkan had followed some of his patients as long as ten years. No late complications have been observed by us. The operation, therefore, tends to circumvent the objections to the various filtration procedures, such as late infection and cataractous changes.

SUMMARY

1. The tension in 11 of 14 eyes with congenital glaucoma was successfully lowered by goniotomy.

2. Failure occurred only in the eyes in which the disease was advanced.

3. Goniotomy failed to control the tension in the two eyes of the same patient in which the elevated tension was associated with bilateral port wine nevus of the face and eyelids.

4. Goniotomy seems to be a fairly simple and safe procedure.

5. All the patients successfully treated by goniotomy have been followed two years or longer.

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OPERATIONS FOR BLEPHAROPTOSIS

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LOS ANGELES

A DROOPING lid that covers a normal eye renders a good eye useless, disfigures a face and shuts in a personality.

The problem of ptosis looms before the ophthalmologist at various times and must be faced. The surgical procedure used depends on the type of training (ophthalmologist or plastic surgeon) and where the training was obtained. The ophthalmologist uses one of the many procedures for correction of the ocular muscles, whereas the plastic surgeon usually prefers one utilizing the occipitofrontalis muscle. Constantly good end results, like those in every other routine, depend on frequent practice and experience. Improvements in technics have continued to be made; in my opinion, one procedure (the Dickey method) offers constantly good end results even for the occasional operator if the various steps are faithfully followed. When the ptosis is of recent origin, less than one year, one is chiefly concerned with finding the cause. When the ptosis is of several years' duration, one is chiefly concerned with surgical correction, though the cause is not entirely forgotten.

There are two general types of ptosis, either of which may be partial or complete, unilateral or bilateral: congenital and acquired. Both the congenital and the acquired type may be further subdivided into (*a*) true ptosis and (*b*) pseudoptosis.

CAUSES OF PTOSIS

The causes of congenital ptosis are: (*a*) weakness of the levator palpebrae superioris (striate) muscle, or Müller's (smooth) muscle of the lid, due to progressive muscular dystophy; (*b*) disorder of a peripheral nerve to the levator, or (*c*) disorder of the central nervous system.

Causes of acquired ptosis are usually intoxications, either acute or subacute: (*a*) syphilis or leprosy involving the roots, the nucleus, the supranuclear centers or the cortical centers of the third nerve; (*b*) lesions of the brain substance, such as hemorrhage, abscess formation or tumor; (*c*) various diseases of the nervous system, such as myas-

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thenia gravis and multiple sclerosis, and (*d*) trauma, accidental or surgical. In cases of the acquired type due to intoxications, surgical intervention should not be contemplated sooner than one year.

Causes of pseudoptosis are (*a*) abnormal widening of the opposite palpebral fissure [1]; (*b*) disease or increased weight of the lid, such as a trachomatous infiltration, tumors of the lid, spasm of the orbicularis muscle and neurofibromatosis; (*c*) paralysis of the superior rectus muscle of one eye with spasm of the inferior oblique muscle of the affected eye, causing the cornea to turn up under the lid, with production of an apparently severe ptosis of one eye when no ptosis is present [2]; (*d*) ptosis which is bilateral but of the outer half of the eye only.

CLINICAL CLASSIFICATION OF TRUE PTOSIS [2]

True ptosis may be classified clinically as follows:

Type 1: Unilateral ptosis without involvement of the homolateral superior rectus muscle.

Type 2: Unilateral ptosis with involvement of the homolateral superior rectus muscle.

Type 3: Bilateral ptosis without involvement of the superior rectus muscles.

Type 4: Bilateral ptosis with involvement of the superior rectus muscles.

Type 5: Unilateral ptosis with weakness of both superior rectus muscles, more pronounced in the homolateral eye.

Type 6: Ptosis with more or less severe paralysis of the third nerve and even of the sixth nerve.

Type 7: Ptosis with the classic jaw-winking reflex, the Marcus Gunn syndrome of misdirection of the developing fifth cranial nerve and oculomotor nerve fibers.

Type 8: Ptosis with the Duane retraction syndrome.

Type 9: Ptosis with neurofibromatosis.

CORRECTION OF PTOSIS

For cosmetic reasons, and because of the mental strain produced by the facial disfiguration of ptosis, the condition should be corrected, by surgical means or otherwise. Secondary deformities or effects of ptosis of great import are (*a*) amblyopia, due to obstructed vision;

1. Meyer, S. J.: Diagnosis and Treatment of Blepharoptosis, Illinois M. J. **91**:89-92 (Feb.) 1947.

2. Spaeth, E. B.: Ptosis, Congenital, Classification: Principles of Surgical Correction, Tr. Am. Acad. Ophth. (1942) **47**:285-301 (March-April) 1943.

(*b*) change in curvature of the spine[3]; (*c*) torticollis; (*d*) spasm of occipitofrontalis muscle, and, last, but not least (*e*) a feeling of inferiority.

The nonsurgical method of correction of ptosis is use of a crutch glass (attached to the spectacles) for the drooping lid. This is for the patient who refuses operation or for some medical reason (anesthesia of the cornea; complete palsy of the nerve) cannot undergo surgical treatment.

The operations for ptosis comprise less than 1 per cent of all surgical procedures on the eye and its appendages.[1] To raise the lid so that it resembles a normal lid, so opening the patient's personality to the outside world and revealing the glint in his eye, is an incomparable satisfaction to the surgeon for work well done and skill attained.

Before an attempt at surgical correction is made, data should be obtained on the following:

(1) Vision in the two eyes and the presence of amblyopia, diplopia or fusion; (2) the sensibility of the cornea, as an estimate of its ability to withstand exposure; (3) the vertical width of the palpebral fissure with the eyes looking straight ahead, with the eyes looking up and with the eyes looking down; (4) estimation of the strength of the elevator power of the muscles of the eyelid when acting alone by computing the measurements in (3) with the patient employing the aid of the frontalis muscle, which arches the brow and corrugates the forehead, with the measurements when the brow is depressed by the fingers of the observer; (5) exophthalmometric reading, if proptosis is suspected; (6) extraocular motility of each eye, especially that of the superior rectus muscle, and the fields of fixation, and (7) effect of cocaine on the smooth muscle of the lid.

The four basic surgical methods for correction of ptosis are (1) shortening of the lid (Kuhnt-Heisrath operation), (2) shortening of the levator palpebrae superioris muscle (Blaskovics, 1923), (3) utilization of the superior rectus muscle (Motias, 1898), (4) utilization of the occiptofrontalis muscle (Dransart, 1879). There are many modifications of these four basic operations for the correction of ptosis, their number indicating efforts to achieve a technic with constantly good results.

Surgical correction for the various types of true ptosis are suggested for the occasional operator as follows:

Type 1: Dickey operation for ptosis.

Type 2: Dickey operation (if the inferior oblique of the affected eye is normal and there is no hypotropia. If hypotropia is present,

3. Meek, R. E.: Ptosis: Applied Anatomy of Eye; Relation to Ophthalmic Surgery, Arch. Ophth. **26**:494-513 (Sept.) 1941.

this should first be corrected or the procedure indicated for type 4 carried out.)

Type 3: Dickey operation bilaterally.

Type 4: First choice: Dickey operation after advancement of both superior rectus muscles and recession of both inferior rectus muscles. Second choice: utilization of occipitofrontalis muscle.

Type 5: Dickey operation for ptosis after advancement of the superior rectus muscle on the affected side. Recession of the overacting inferior oblique for relief of diplopia and other indicated surgical correction of extraocular muscles.

Type 6: Crutch glass as first choice, or some utilization of the occipitofrontalis muscle (combined with measures to place the deviating eye in the forward position, such as recession of the external rectus muscle and advancement with resection of the internal rectus muscle, provided no corneal anesthesia is present).

Type 7: Dickey operation.

Type 8: Recession of the internal rectus muscle, followed by the Dickey operation if correction of ptosis is insufficient. The first procedure frequently corrects the retraction syndrome and thereby greatly improves the ptosis.

Type 9: Utilization of the occipitofrontalis muscle after plastic surgical procedures.

Kuhnt-Heisrath Operation.—This procedure consists in excision of a great part of the tarsus, in an effort to lighten the weight of the lid, as in cases of trachoma.

Blaskovics Operation.—There is no absolute means of measuring in millimeters the amount of muscle to be shortened in order to correct a given degree of ptosis, but one can follow the rule [4] that removal of about 2 mm. of muscle or of about 1 mm. of cartilage is required to correct 1 mm. of ptosis. Thus, considering a normal width of fissure as about 11 mm., if there is a deficiency in width of about 6 mm., 12 mm. of muscle must be removed to secure a width of fissure of 11 mm.

The mere fact that there are no absolute means of measuring the amount of levator muscle to be shortened, or the strength of the weakened levator, permits an uncontrolled variable to be ever present, so that, in the hands of most operators, the results of the Blaskovics operation cannot be depended on.[5] Undercorrection is the rule, although occasional overcorrection in the lateral half occurs.

4. Modified Blaskovics Operation for Ptosis, Bull. Pract. Ophth. **12**:7-9 (Jan.) 1942.

5. Fink, W. H.: Surgical Management of Ptosis, Journal-Lancet **60**:245-246 (May) 1940.

Motais Operation.—Most surgeons insist that the action of the superior rectus be strong[4]; yet the procedure may also be successful when the superior rectus is partially paralyzed, provided there is no hypotropia and the inferior oblique is normal. If both the superior rectus and the inferior oblique are paralyzed, this operation is not indicated. It is usually contraindicated in cases of unilateral ptosis, as there exists the danger of producing postoperative hypotropia and diplopia. If the cornea is anesthetic, complications may arise, as in sleep the lower part of the cornea is always exposed after this operation. However, when successfully performed, the Motais operation provides for the synchronous movement of the upper lid with that of the globe in looking up and down. Good elevation of the lid is secured, and a satisfactory lid fold develops. An important criticism of this operation is the resultant notching of the upper lid, which may necessitate a graduated tarsectomy at the time of the original operation, or later. Another important reason for failure is that the sutures employed in attaching the tongue of the superior rectus muscle to the tarsus may pull out or the adhesions produced between the upper lid and the globe may pull out or become stretched. If the entire width of the superior rectus muscle is attached to the lid without severing its tendinous attachment to the globe, symblepharon with entropion and districhiasis results.

Diplopia may result if the superior rectus muscle is weakened too much. However, if only the middle third of the muscle is used, the small amount of diplopia produced will not be annoying. A disturbance in the coordination of the act of closure of the lids results, and the globe is prevented from rolling up normally.

A study of the extraocular muscles should be made in all cases, as there is often a weakness of the elevator muscles of the globe in association with ptosis. Such a hypotropia should first be remedied before any surgical correction of the ptosis is attempted, especially if one contemplates utilization of the superior rectus muscle to lift the ptosed lid. Any exotropia or esotropia should be corrected first before operation for ptosis is done.

The use of a fascia lata sling attached to each end of the tarsus and running under the superior rectus muscle has come into favor in recent years (Dickey operation); this procedure removes many of the disadvantages of the Motais operation. However, the innervation, origin and course of the muscle fibers of the levator and the superior rectus are the same, so that no reeducation is required for the new function of the muscle. The easiest approach in this operation is through the conjunctiva.

One should note the position of the upper lid fold of each eye before operation, so that the new lid fold may be placed in its proper place at the time of operation.

Dransart Operation.—This procedure, which consists in the production of adhesions between the frontalis muscle and the upper lid, although easy of accomplishment, is not good surgery. It leads to a straigth upward pull on the lid and accentuates the already present smooth lid effect. The implantation of deep long sutures and buried strips of epidermis is open to serious objections. If all the elevators of the globe and lid are paralyzed, it may be necessary to use fascia lata or fibers of the orbicularis. While such an operation probably

A-G, results of the Dickey operation for ptosis. *A*, *C* and *E* are preoperative photographs; *B*, *D* and *F* show postoperative results.

A (case 1, age 29), severe bilateral congenital ptosis, showing extended chin, elevated eyebrows and corrugated forehead, timidity and inferiority complex.

B (case 1), correction of ptosis by the Dickey operation. Note absence of the elevated brows, corrugated forehead and extension of the chin. The lids moved up and down like normal lids.

C (case 2; age 49), severe bilateral acquired ptosis, of undetermined cause, more pronounced on the left side.

D (case 2), correction of ptosis by the Dickey operation (bilateral). The glint in the eyes is there.

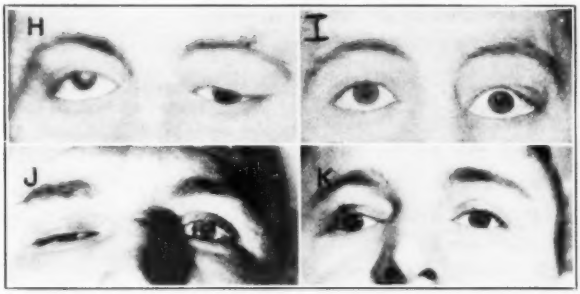
E (case 3; age 24), very severe unilateral congenital ptosis.

F (case 3), correction of unilateral ptosis by the Dickey operation.

G (case 3), postoperative photograph, showing that the natural, synchronous upward movements of the lids.

constitutes the type of surgical correction most frequently performed, the end results are usually far from satisfactory. If the lower, or lid, attachment of the implantation is in the middle third of the tarsus, a postoperative notching may develop, which is unsightly and may be corrected later only by a tarsectomy. Hence the lid attachment of the implantation should be at the junction of the middle and the inner third, as this is usually the highest part of the upper lid and attachment here gives a better cosmetic result.

If diplopia is present before operation when the ptosed lid is raised, an attempt should be made to correct it before correction of the ptosis. If the ptosed eye is amblyopic, the danger of disturbing fusion or of producing postoperative diplopia is negligible. If the tactile sensibility of the cornea is lost, one can be almost certain that uncovering the cornea will sooner or later result in neuroparalytic keratitis, with its serious consequences.



H-K, results of the Weiner-Alvis modification of the operation utilizing the occipitofrontalis muscle. *H* and *J* are preoperative, and *I* and *K* postoperative, photographs.

H (case 4; age 22), severe unilateral congenital ptosis and concomitant hypotropia.

I (case 4), correction of hypotropia and correction of ptosis by modified Dransart operation, using fascia lata. Results are fair, but not as good as with the Dickey operation in case 3. The corrected left lid has flat appearance and an unsightly fold.

J (case 5; age 25), severe ptosis and concomitant severe unilateral congenital hypotropia.

K (case 5), correction of ptosis and hypotropia by a modified Dransart operation, using fascia lata. Results are fair but not as excellent as in case 3. The affected lid does not move up and down like its fellow, but must be raised by elevating the brow.

Dickey Operation for Ptosis.—The many surgical procedures for the correction of ptosis indicates that satisfactory results cannot always be depended on, even in the best of hands. Because of the relatively rarity

of this condition, most surgeons do not have the opportunity to perform many operations to improve their technic, and the search for one procedure which can be depended on for satisfactory end results in most cases continues, as new improvements are tried. I have heard several eminent ophthalmologists in various parts of the country state that they use the Blaskovics procedure in all cases of ptosis. Plastic surgeons say they use some modification of the Dransart technic in all cases of ptosis. It is my opinion that the Dickey (modified Motais) operation will give constantly excellent results in any type of ptosis except as noted later in the contraindications, in which case a Dransart operation [6] or one of its modifications is used. The technic of the Dickey operation can be obtained elsewhere.[7] The Dickey operation for ptosis has the following advantages:

1. The corrected height of the lid can be depended on to remain where it is fixed at the operating table. No overcorrection or undercorrection, which may not adjust itself after operation, is necessary.

2. The operation is not difficult to perform (the conjunctival route is easier than the external, or skin, route).

3. Neither the superior rectus muscle nor any portion of it is detached from the globe.

4. The whole muscle, rather than the fibers of its middle third only, lends itself to the support of the lid.

5. There is a minimum chance of any sutures pulling loose.

6. It produces a firm attachment between the superior rectus muscle and the upper lid.

7. The cosmetic and functional results are very good in suitable cases.

8. A wide palpebral fissure, or one resembling the unaffected side, may be secured, as desired.

9. The fissure resembles the normal elliptic ones, and the lid fold is usually satisfactory.

10. The lid follows the eye in its vertical movements.

11. There is no contraindication, no diplopia of any importance in cases of unilateral ptosis provided the superior rectus of the affected side is normal.

12. If, for any reason, the operation must be undone, this can be achieved in the office simply by everting the lid and cutting one arm of the fascia.

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6. Rosenburg, S.: Ptosis: Fascia Lata Transplant, Am. J. Surg. **47**:142-148 (Jan.) 1940.

7. Cordes, F. C., and Fritschi, U.: Dickey Operation for Ptosis: Results in Twenty-One Patients and Thirty Lids, Arch. Ophth. **31**:461-468 (June) 1944.

The dressing is an important part of the technic. The Friedenwald procedure, in which a lower lid suture is inserted to pull the lower lid upward over the cornea (to avoid exposure keratitis) and lend temporary support to the upper, or corrected, lid is excellent.[8]

Contraindications to the Dickey operation for ptosis are (1) anesthesia of the cornea, (2) paralysis of the superior rectus and inferior oblique of the affected eye, (3) neurofibromatosis and (4) complete paralysis of the third nerve.

Without a doubt, any operative procedure which corrects a condition so that a resemblance to the normal structure and function is established is the one of choice. In accordance with this precept, the Blaskovics operation would be foremost if the operator could depend with certainty on his results. Otherwise, the results of the Dickey operation, though not restoring the normal structure, can be depended on even by the occasional operator and gives definitely better cosmetic and function results than utilization of an occipitofrontalis muscle or other procedures.

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8. In a child aged 14 months, for whom pictures were not available, severe unilateral congenital ptosis, producing head tilt to the right shoulder and extension of the chin toward the left shoulder, was corrected by the Dickey operation, with alleviation of the ptosis, the head tilt and the rotation of the chin. The corrected lid now moves up and down synchronously with its unaffected fellow.

Ophthalmologic Reviews

NEUROMYELITIS OPTICA (DEVIC'S DISEASE)

Presentation of Five Cases, with Pathologic Study, and Review of Literature

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SUDDEN, complete binocular loss of vision in a healthy young adult is undoubtedy a major catastrophe to the patient. When examination of the afflicted person reveals no pathologic basis for the blindness, it becomes a problem of major importance to the ophthalmologist. Such is often the case in the rare syndrome called neuromyelitis optica, or Devic's disease.[1] This condition is commonly ushered in by sudden, total blindness, followed by a severe, rapidly ascending inflammation of the spinal cord, terminating in death. The cause of this malady is unknown, and there is no known method of effective therapy. Pathologically, acute necrosis of nerve tissue is observed, principally in the optic nerves and the spinal cord. Histologically, destruction of myelin sheaths, axis-cylinders, nerve cells and neuroglia is seen, accompanied with infiltration of white blood cells and with a feeble attempt at repair by the nerve tissue.

Neuromyelitis optica is but one of the group of so-called demyelinating diseases of the central nervous system which exhibit this pathologic picture. The other components of this group are: (1) multiple sclerosis, the commonest; (2) the acute disseminated encephalomyelitides that follow vaccination, foreign serum therapy and the acute infectious diseases of children, and (3) the diffuse scleroses, including Schilder's disease (progressive subcortical encephalopathy), Pelizaeus-Merzbacher disease (aplasia axialis extracorticalis congenita), Krabbe's disease (infantile familial diffuse cerebral sclerosis, Scholz's disease (juvenile familial diffuse cerebral sclerosis) and Baló's disease (an infantile demyelinating encephalopathy). These are all acute processes with a high mortality rate, with the exception of multiple sclerosis. They are all commonest in the first half of the average life, multiple sclerosis and neuromyelitis optica occurring usually in young adults and the others oftenest in children. The clinical picture varies with

1. This syndrome is also known as myelitis with optic neuritis (Dreschfeld), optic neuritis and myelitis (Sharkey), neuroptic myelitis (Devic), ophthalmo-neuromyelitis (de Lapersonne), ophthalmo-encephalo-myelopathy (Barrera) and optic encephalomyelitis (Klar). In addition to the Latin term, neuromyelitis optica, the condition is frequently called optic neuromyelitis or, more simply, neuromyelitis.

the location of the lesions in the nervous system but always shows a decided trend toward involvement of the optic pathway. Microscopically, the lesions of all these syndromes are similar. The differential diagnosis of the various types is a matter of standing dispute, largely because there is no generally accepted terminology. In the absence of knowledge of their etiology, these diseases have been designated by names suggestive of the anatomic distribution or histologic characteristics of their lesions. Pathologic classification fails, because they merge into one another in such a way as apparently to constitute different stages of a single disease process. Clinical classification is unsuccessful, for the same reason; it does not include the transitional types that exhibit features common to two or more pathologic varieties. The only available classification today attempts to correlate both the clinical and the pathologic characteristics and to include the transitional forms. Future investigation may disclose that these clinicopathologic distinctions do not actually correspond to differences in etiology, but that they are the result of differences in severity of the process, the heredity of the patient, the age of the patient, the nature of the precipitating events and the constitution and immunity of the patient. As a group, the demyelinating diseases embody one of the outstanding unsolved problems in the field of medicine.

The preceding paragraphs may suggest the reasons for the ensuing detailed discussion of a rarity like neuromyelitis optica: 1. Although generally considered a rare and unimportant disease, it assumes a new importance if it is a form of multiple sclerosis, as is often proposed today. With its high mortality rate, neuromyelitis optica furnishes more material for pathologic study than multiple sclerosis. 2. More important, because neuromyelitis optica is often fatal in the early acute stage, its study may fill an important void in the pathology of multiple sclerosis. 3. Even though the two diseases are not identical, the study of neuromyelitis optica is worth while because of their close similarity. 4. The fact that the optic apparatus is always involved warrants the attention of the ophthalmologist. 5. Finally, because the subject of the place of neuromyelitis optica among the demyelinating diseases has not been reviewed in the English ophthalmologic literature since 1927, it is time for a reevaluation in the light of recent neuropathologic developments.

HISTORICAL REVIEW

Earlier Literature.—Convinced of the value of ophthalmoscopic study in the diagnosis of lesions of the brain, Allbutt [2] a physician at the Leeds Infirmary in the last century, decided to investigate the

2. Allbutt, T. C.: On the Ophthalmoscopic Signs of Spinal Disease, Lancet **1**:76-78, 1870.

fundus in cases of disease of the spinal cord. Cognizant of the fundus changes in tabes dorsalis, he looked instead for patients with acute processes, either traumatic or inflammatory. In 1870 he published his results in the *Lancet*: (1) In 17 cases of acute trauma to the spinal cord no pathologic change in the fundus had been noted, and (2) of 5 cases of acute nontraumatic myelitis, optic neuritis had appeared twelve weeks after the spinal symptoms in 1. Subsequent authors have considered the last case the first instance of neuromyelitis optica reported in the literature. With no autopsy, Allbutt was unable to elucidate the strange combination of lesions in the optic nerve and spinal cord, but he hypothesized an extension of meningitis from the cord to the base of the brain, and thence to the optic nerves.

The honor of the first thorough clinical description of neuromyelitis optica must be awarded to Erb,[3] who in 1879 reported a case of "myelitis transversa dorsalis with neuritis descendens opticorum." The course in his case was prolonged, but the patient eventually recovered both vision and mobility of the extremities. One year later Seguin [4] presented 2 cases of "optic neuritis and subacute transverse myelitis" before the New York Neurological Society; his patients also survived, after a protracted siege. Both Erb and Seguin expressed the opinion that the coincidence of disease of the optic nerves and that of the spinal cord was an accident, with no causal or pathologic relation. In 1882 Chisholm [5] published the first case report of this syndrome in the American ophthalmologic literature. Achard and Guinon [6] in 1889 published the first pathologic study of neuromyelitis optica, describing the complete disappearance of myelin sheaths in the optic nerves of their patient.

In 1894 Devic [7] summarized the reported cases before the French Congress of Medicine at Lyon and coined the terms *neuro-myélite* and *neuroptico-myélite* for this optic neuritis (he included both papillitis and retrobulbar neuritis) accompanied with acute myelitis and occa-

3. Erb, W.: Ueber das Zusammenvorkommen von Neuritis optica und Myelitis subacuta, Arch. f. Psychiat. **10**:146-157, 1880.

4. Seguin, E. C.: On the Coincidence of Optic Neuritis and Subacute Transverse Myelitis, J. Nerv. & Ment. Dis. **7**:177-188, 1880.

5. Chisholm, J. J.: An Obscure Case in Nerve Pathology Accompanying Optic Neuritis, Arch. Ophth. **11**:239-242, 1882.

6. Achard, C., and Guinon, L.: Sur un cas de myélite aiguë diffuse, avec double névrite optique, Arch. de méd. expér. et d'anat. path. **1**:696-710, 1889.

7. Devic, M. E.: Myélite aiguë dorso-lombaire avec névrite optique: Autopsie, Congrès français de Médicine, Première Session, Lyon, 1894, **1**:434-439, 1895; abstracted, Myélite subaiguë compliquée de névrite optique, Bull. méd., Paris **8**: 1033, 1894.

sionally with other neurologic symptoms. He found 16 cases[8] in the literature, with 4 deaths, and added a fatal case of his own, with autopsy. Discarding the various theories previously advanced to explain the pathogenesis of this syndrome, Devic admitted that nothing was known of either the etiology or the pathogenesis of these anatomically separated lesions, and he stressed the importance of a complete ophthalmologic examination as an approach to the solution. Devic's case report and pathologic observations were expanded into a doctoral thesis by one of his pupils, Gault.[9] Since that time the syndrome has frequently been called Devic's disease.

Survey of the literature on neuromyelitis optica by Goulden[10] in 1914 disclosed 51 case reports,[11] his own making the fifty-second

8. Devic reviewed the following reports: (*a*) Abadie, M.: De l'atrophie des nerfs optiques dans le mal de Pott, Bull. Soc. de chir. **1**:26-32, 1876. (*b*) Steffan, P.: Beitrag zur Lehre des Zusammenhanges der Erkrankungen der Sehnerven mit denen des Rückenmarkes, Ber. d. Ophth. Gesellsch. **17**:90-105, 1879. (*c*) Erb.[3] (*d*) Seguin.[4] (*e*) Chauvel, J.: Névrite optique double avec myélite aiguë temporaire, Bull. et mém. Soc. de chir. de Paris **6**:512-517, 1880. (*f*) Noyes, H. D.: Acute Myelitis mit doppelseitiger Neuritis optica, Arch. f. Augenh. **10**:331-337, 1881. (*g*) Rumpf, T.: Zur Wirkung des faradischen Pinsels bei einem Fall von Neuritis optica und Myelitis transversa, Deutsche med. Wchnschr. **7**:442-443, 1881. (*h*) Dreschfeld, J.: On Two Cases of Acute Myelitis Associated with Optic Neuritis, Lancet **1**:8-9 and 52-53, 1882. (*i*) Sharkey, S. J.: Embolism of the Right Middle Cerebral Artery Producing Left Hemiplegia and Hemianesthesia: Absorption of a Large Portion of the Right Hemisphere; Death Seven Years Later, M. Times & Gaz. **1**:846-847, 1884. (*j*) Knapp, H.: Ein Fall von acuter Myelitis mit beiderseitiger Ophthalmoplegia und Stauungspapille berichtete, Klin. Wchnschr. **22**:835-836, 1885. (*k*) Firth, R.: Double Optic Neuritis with Paralysis of One Arm, Following an Injury to the Spine, Practitioner **36**:426-429, 1886. (*l*) Achard and Guinon.[6] (*m*) Drake-Brockman, E. F.: A Case of Double Optic Neuritis Dependent on Spinal Disease, Brit. M. J. **2**:77-78, 1892. (*n*) Fuchs, S.: Klinische und anatomische Untersuchungen über einen Fall von multipler Neuritis mit Erkrankung der NN. optici, Deutsche Ztschr. f. Nervenkrank. **4**:38-78, 1893. (*o*) Schanz, F.: Ueber das Zusammenvorkommen von Neuritis optica und Myelitis acuta, Deutsche med. Wchnschr. **19**:615-617, 1893.

9. Gault, F.: De la neuromyélite optique aiguë, Thesis for the Doctorate, Lyons, no. 0981, 1894, p. 104.

10. Goulden, C.: Optic Neuritis and Myelitis, Tr. Ophth. Soc. U. Kingdom **34**:229-252, 1914.

11. In addition to the papers previously discussed by Devic, Goulden reviewed the following publications: (*a*) Dedone, P.: Étude sur la névrite optique, Thesis, Paris, no. 202, 1875, p. 50. (*b*) Thorowgood, J. C.: Case of Optic Neuritis, with Complete Loss of Vision: Recovery under Treatment, Tr. Clin. Soc. London **8**:80-83, 1875. (*c*) Putzel, L.: Remarks on a Case of Neuritis with Secondary Inflammation in the Spinal Cord, M. Rec. **15**:390-392, 1879. (*d*) Rieger, F., and Forster, R.: Auge und Rückenmark, Arch. f. Ophth. **27**:109-202, 1881. (*e*) Schluter, F.: Ueber Neuritis optica, Inaug. Dissert., Berlin, G. Schode, 1882, p. 42. (*f*) Sharkey, S. J., and Lawford, J. B.: On a Case of Acute Optic Neuritis

(Footnote continued on next page)

reported case. Goulden claimed that his patient, a white man aged 60, was older than any of the preceding patients; the patient suffered nearly complete loss of vision in six days, followed by severe ascending myelitis and death sixteen days after onset of the visual difficulty. According to Goulden's survey, blindness preceded the spinal syndrome in over 80 per cent of the cases. Ophthalmoscopically, optic neuritis (ranging from slight hyperemia of the disk to full-blown edema with dilated, tortuous veins, hemorrhages and exudates) was seen; retrobulbar neuritis was rare. He found nothing characteristic about the myelitis; the symptomatology was that of ordinary acute ascending inflammation of the cord. Goulden stressed the similarity of the course of the myelitis and the optic neuritis; both showed slight symptoms in the beginning, both progressed to a period of great intensity and, if death did not supervene, both retrogressed to complete, or almost complete, recovery. From his study of the pathology, Goulden decided that the two processes, visual and spinal, were due to a common infectious cause.

Associated with Acute Myelitis, Tr. Ophth. Soc. U. Kingdom **4**:232-243, 1884. (*g*) Knapp, H.: Ueber einen Fall von acuter Myelitis mit beiderseitiger Ophthalmoplegia und Stauungspapille, Neurol. Centralbl. **4**:502-503, 1885. (*h*) Picqué, L.: Étude critique sur l'anatomie pathologique et al pathogénie des névrites optiques, Arch. d'opht. **18**:420-435, 1888. (*i*) Eskridge, J. T.: Acute Myelitis Preceded by Acute Optic Neuritis, J. Nerv. & Ment. Dis. **17**:609-615, 1890. (*j*) Elschnig, A.: Klinischer und anatomischer Beitrag zur Kenntniss der acuten retrobulbären Neuritis, Arch. f. Augenh. **26**:56-80, 1893. (*k*) Mahokian, P. A.: Neuritis optica bei Myelitis acuta, Inaug. Dissert., Berlin, O. Francke, 1893, p. 30. (*l*) Dreschfeld, J.: Acute Disseminated Myelitis, Brit. M. J. **1**:1174-1177, 1894. (*m*) Hoffman, J.: Ueber das Zusammenvorkommen von Sehnerven- und Rückenmarksentzündung, Neurol. Centralbl. **15**:671-672, 1896. (*n*) Katz, K.: Ueber das Zusammenvorkommen von Neuritis optica und Myelitis acuta, Arch. f. Ophth. **42**:202-240, 1896. (*o*) Taylor, F.: Myelitis and Optic Neuritis, Guy's Hosp. Rep. **53**:45-54, 1896. (*p*) Schuster, P., and Bielschowsky, M.: Beitrag zur Pathologie und Histologie der multiple Sklerose, Ztschr. f. Klin. Med. **34**:395-418, 1898. (*q*) Dalen, A.: Neuritis optica und Myelitis acuta, Arch. f. Ophth. **48**:672-693, 1899. (*r*) Schuster, P., and Meindel, K.: Neuritis optica als Complication bei Erkrankungen des Nervensystems, Neurol. Centralbl. **18**:1018-1023, 1899. (*s*) Taylor, J., and Collier, J.: The Occurrence of Optic Neuritis in Lesions of the Spinal Cord: Injury, Tumour, Myelitis: An Account of Twelve Cases and One Autopsy, Brain **24**:532-554, 1901. (*t*) Faure, A.: De la neuromyélite optique aiguë, Thesis, Lyons, no. 45, 1903, p. 66. (*u*) Weill, M., and Gallavardin, M.: Sur un cas de neuromyélite optique aiguë, Lyon méd. **101**:207-209, 1903; (*v*) Note sur l'anatomie pathologique de la myélite aiguë diffuse (myélites a cellules épithélioides), Rev. neurol. **11**:999-1003, 1903. (*w*) Brissaud, E., and Brécy, N.: Neuromyélite optique aiguë, ibid. **12**:49-54, 1904. (*x*) Kerschensteiner, N.: Ueber Neuromyelitis optica, Med. Wchnschr. **53**:802-805, 1906. (*y*) Hillion, H.: De la neuromyélite optique aiguë, Thesis, Paris, no. 214, 1907, p. 160. (*z*) de Lapersonne, F.: La syndrome de la névrite optique associée a la myélite ophthalmoneuro-myélitis, Rev. neurol. **21**:378-381, 1911.

In 1927 the literature on neuromyelitis optica was again surveyed by Beck,[12] who found the diagnosis confused by the inclusion of coexistent myelitis and papillitis due to other causes; he noted that this combination may be seen in influenza, poliomyelitis, tumors and injuries of the cord, postencephalitic conditions and disturbances of lactation. Beck found multiple sclerosis and Schilder's disease most frequently confused with neuromyelitis optica. Including only those cases in which the principal symptoms were paraplegia and blindness, Beck[13] unearthed reports of 70 cases. According to his findings, the myelitis more often preceded the optic neuritis. Pathologic examinations were made in 25 of the cases in Beck's series; in 9 cases the lesions of the cord consisted of disseminated foci, while in 16 there was diffuse myelitis, varying in extent from 1½ inches (3.8 cm.) to nearly all the cervical, thoracic and lumbar segments. Beck suggested that these two pathologic types may represent two distinct diseases with a common clinical picture. His patient, a 15 year old girl, had four attacks of the disease, from three of which she made a complete recovery; then came a final loss of vision with a rapidly ascending paralysis that ended in blindness and death. Beck stated that the three remissions made his case unique; in his series he came across 7 instances of transient improvement for a few days, but no definite remissions such as these. Beck, too, concluded that neuromyelitis optica is a specific disease entity.

English Literature Since 1927.—There have been a number of case reports but no complete review of neuromyelitis optica in the opthalmo-

12. Beck, G. M.: A Case of Diffuse Myelitis Associated with Optic Neuritis, Brain **50**:687-703, 1927.

13. Beck reviewed the following articles, in addition to most of the works reviewed by Devic[8] and Goulden[11]: (*a*) Fürstner, N.: Zur Kenntniss der acuten disseminierten Myelitis, Neurol. Centralbl. **18**:155-161, 1899. (*b*) Ulrich, G.: Beobachtung von Opticuserkrankungen akuter genuiner Myelitis und Polyneuritis, Ztschr. f. Augenh. **29**:195-196, 1913. (*c*) Abelsdorf, G.: Akute retrobulbäre Sehnervenentzündung bei Myelitis mit Sektionsbefund, Ztschr. f. Klin. Med. **85**: 435-442, 1918. (*d*) Henneberg, G.: Ueber einem Fall von Myelitis cervicalis mit Opticuserkrankung und Brown-Séquardscher Lähmung, Neurol. Centralbl. **37**: 223-224, 1918. (*e*) Karplus, J. P.: Organische, nichttraumatische Nervenkrankheiten bei Kriegsteilnehmen, Wien. med. Wchnschr. **9**:137-148, 1919. (*f*) Buchanan, L.: Monocular Optic Neuritis, Brit. J. Ophth. **7**:170-174, 1923. (*g*) Jumentié, M. M., and Valière-Vialeix, V.: Sur un cas d'encéphalite aiguë non-suppurée de l'adulte avec névrite optique: Aspect ophtalmoscopique de stase papillaire; lésions à localisations prédominant sur le trajet des voies optiques intra et extra cérébrales, Ann. d'ocul. **162**:14-41, 1925. (*h*) Jendralski, F.: Die Entzündung des Sehnerven bei Myelitis acuta, Klin. Monatsbl. f. Augenh. **71**:19-28, 1923. (*i*) Genet, F., and Devic, C.: Neuromyélite optique aiguë persistence anomale de séquelles neurologiques et oculaires, Ann. d'ocul. **162**:488-489, 1925. (*j*) Bouchut, L., and Dechaume, J.: Étude histopathologique d'un cas de neuropticomyélite aiguë, Ann. d'anat. path. **4**:357-372, 1927.

logic literature since 1927. In 1933 McAlpine [14] described a case in which the clinical diagnosis was disseminated sclerosis but at autopsy the diagnosis was changed to neuromyelitis optica. Cone, Russel and Harwood [15] reported a case in 1934 in which lead was demonstrated in the tissues of the brain and cord; they suggested this metal as the causative factor of the disease. Berliner [16] in 1935 discussed multiple sclerosis, acute disseminated encephalomyelitis, neuromyelitis optica and encephalitis periaxialis diffusa together as the demyelinating diseases of the nervous system and agreed with others that they may be variations of one entity, with clinical differences due to varying degrees of involvement or to predilection for special tissues. Walsh [17] in 1935 published the clinicopathologic study of a patient with neuromyelitis optica and the clinical histories of 3 other patients who survived. The first patient was a 9 year old girl who suffered blindness and ascending motor and sensory paralysis and died six weeks after the onset of the process. For 2 of the other patients an erroneous diagnosis of brain tumor was made and an exploratory operation performed, followed by almost immediate death. Walsh concluded that exploratory craniotomy or lumbar puncture in the demyelinating disease may cause an exacerbation of the process. In regard to specificity of Devic's syndrome, he concluded that his and similar cases are sufficiently distinctive clinically to warrant a special designation.

Balser [18] in 1936 discussed 3 cases of neuromyelitis optica pathologically and a fourth case clinically. Citing the work of Hurst,[19] Balser stated the opinion that it is unlikely that this disease is caused by a virus but he did concede the possibility in view of the small knowledge of virus diseases. He found remissions in a disease of such severity surprising. Hassin [20] published a case report in 1937 with a thorough pathologic study. He compared neuromyelitis optica with multiple sclerosis and disseminated encephalomyelitis and concluded that, although it might be impossible to differentiate these conditions clinically, it can be done microscopically. Fralik and

14. McAlpine, D.: Myelitis with Blindness: Acute Disseminated Encephalomyelitis, Proc. Roy. Soc. Med. **27**:662-664, 1933-1934.

15. Cone, W. V.; Russel, C., and Harwood, R. U.: Lead as a Possible Cause of Multiple Sclerosis, Arch. Neurol. & Psychiat. **31**:236-269 (Feb.) 1934.

16. Berliner, M.: Acute Optic Neuritis in Demyelinating Diseases of the Nervous System, Arch. Ophth. **13**:83-98 (Jan.) 1935.

17. Walsh, F. B.: Neuromyelitis Optica, Bull. Johns Hopkins Hosp. **56**:183-210, 1935.

18. Balser, B. H.: Neuromyelitis Optica, Brain **59**:353-365, 1936.

19. Hurst, E. W.: The Neurotropic Virus Diseases: Wuthering Lecture of 1935, Lancet **2**:697-702, 1935.

20. Hassin, G. B.: Neuroptic Myelitis Versus Multiple Sclerosis: A Pathologic Study, Arch. Neurol. & Psychiat. **37**:1083-1099 (May) 1937.

DeJong,[21] in reporting the disease in a young lawyer, with acute loss of vision and an ascending spinal paralysis, progressing to death in six weeks, admitted the similarity of this process to multiple sclerosis, disseminated encephalomyelitis and diffuse sclerosis but stated that it could be differentiated clinically and pathologically from these other demyelinating diseases. Dolgopol [22] reported a case that was uncommon in two respects, namely, that the patient was a Negro and that she lived three years and eight months before succumbing to the disease.

McKee and McNaughton [23] described 2 cases in 1938, in both of which recovery occurred. One of their patients was followed for five years after recovery and visual acuity, visual fields and fundi remained normal. In addition to the usual signs and symptoms of visual and spinal involvement, McKee and McNaughton found that both their patients showed slight signs of invasion of the brain stem. In the discussion of their paper, Sanford Gifford [24] described 3 cases he had seen. He likened this syndrome to multiple sclerosis and said that in this condition one sees in a few short weeks what happens in multiple sclerosis over many years. Gifford raised the interesting speculation whether the patients who recover will later show signs of multiple sclerosis. Fatal neuromyelitis optica occurring in identical twin sisters was recorded by McAlpine [25] in 1938; 1 of the twins died of the disease at the age of 24 and the other at the age of 26 years. McAlpine collected 22 cases of this disease from the literature and divided them into three categories according to their clinical course: (1) cases which follow a progressive course with death in from five days to several weeks (10 cases); (2) cases which show one or more remissions but the patients finally die after several months (7 cases), (3) cases in which there is partial or complete recovery (5 cases).

In 1940 Fetterman and Chamberlain [26] reported a case of neuromyelitis optica in which there was complete recovery. These authors emphasized the necessity of careful differentiation between optic neuritis and papilledema from increased intracranial pressure. Although there are apparently striking clinical differences between Devic's disease and multiple sclerosis, Fetterman and Chamberlain suggested that the differ-

21. Fralik, F. B., and DeJong, R. N.: Neuromyelitis Optica, Am. J. Ophth. **20**:1119-1124, 1937.

22. Dolgopol, V. B.: Neuromyelitis Optica, with Pathologic Study of a Case, Arch. Neurol. & Psychiat. **39**:561-569 (March) 1938.

23. McKee, S. H., and McNaughton, F. L.: Neuromyelitis Optica: A Report of Two Cases, Am. J. Ophth. **21**:130-137, 1938.

24. Gifford, S., in discussion on McKee and McNaughton,[23] p. 136.

25. McAlpine, D.: Familial Neuromyelitis Optica: Its Occurrence in Identical Twins, Brain **61**:430-448, 1938.

26. Fetterman, J. L., and Chamberlain, W. P., Jr.: Syndrome of Neuromyelitis Optica, Arch. Ophth. **23**:577-583 (March) 1940.

ence may be merely that of the time element, rather than of the etiologic agent or the pathologic picture. Because of the anatomic and physiologic complexity of the nervous system, the same etiologic agent may produce many different syndromes by pure chance of site and tempo of involvement; according to these writers, among the combinations of areas commonly affected (like 7 and 11 on the dice) are the optic nerves and spinal cord.

Utilizing the criteria laid down by Devic in 1894, Putnam and Forster [27] found 12 cases of neuromyelitis optica among the records of the Neurological Unit of the Boston City Hospital and the Neurological Institute of New York. The principal myelitic lesions corresponded to the description given by Devic, but there were, in addition, milder lesions scattered throughout the nervous system. Putnam and Forster attributed the cure in these cases to good treatment: tidal drainage for the cystitis and the Drinker respirator for respiratory failure. They followed 7 patients for six months or longer; 6 patients exhibited new neurologic manifestations and the condition became clearly recognizable as multiple sclerosis. They concluded, therefore, the Devic's syndrome is a subvariety of multiple sclerosis, except in a few bizarre and atypical cases.

In 1945 Kohut and Richter [28] reported 2 cases of neuromyelitis optica, 1 studied clinically and the other, with fatal outcome, with a complete work-up. Their pathologic description in the latter case is excellent. These authors favored the unicity of neuromyelitis optica, concerning which they said:

> . . . the pathologic picture offered by the present case does not conform to even the broadest pathological conception of either multiple sclerosis or disseminated encephalomyelitis. Diffuse, massive necrosis of the spinal cord coupled with acute degeneration of the optic nerves, and complete absence of dissemination elsewhere in the central nervous system, constitute a pathological complex *sui generis*.

In the same year, Silbermann [29] contributed the history of a patient who lived, which was unusual in two respects: (1) The patient was a Negro, and (2) spinal block, substantiated by roentgenographic examination, occurred during the acute phase of the disease.

French Literature.—In 1930 Michaux,[30] studying under Guillain at the Salpêtrière, wrote an excellent doctoral thesis on neuromyelitis

27. Putnam, T. J., and Forster, F. M.: Neuromyelitis Optica: Its Relation to Multiple Sclerosis, Tr. Am. Neurol. A. **68**:20-25, 1942.

28. Kohut, H., and Richter, R. B.: Neur-Optic Myelitis: A Clinico-Pathological Study of Two Related Cases, J. Nerv. & Ment. Dis. **101**:99-114, 1945.

29. Silbermann, S. J.: Devic's Disease: A Clinical Review and Case Report, J. Nerv. & Ment. Dis. **102**:107-120, 1945.

30. Michaux, L.: La neuro-myélite optique aiguë, Thesis, Paris, Lousi Arnette, 1930, p. 289.

optica. He analyzed in detail his case and 21 cases from the literature.[31] Michaux attached no significance to the precedence of the visual or spinal disability; he said that chance determines which shall be first, or whether they come on simultaneously. Clinically, Michaux found neuromyelitis optica characterized chiefly by its variability; he could find no signs or symptoms that could be considered pathognomonic. He decried the fact that the cerebrospinal fluid had been examined in so few cases. Particularly good is Michaux's discussion of the differential diagnosis of this disease; he compared it with multiple sclerosis, cerebrospinal syphilis with optic neuritis, epidemic encephalitis, subacute necrotic myelitis, alcoholic polyneuritis, brain tumor and traumatic lesions of the cervical portion of the spinal cord. Admitting that only the demonstration of the causative agent will be decisive, Michaux concluded that, in the present state of affairs, neuromyelitis optica may be definitely distinguished from other diseases of the nervous system by its pathologic peculiarities.

Marinesco and colleagues [32] in 1930 discussed a fatal case of Devic's syndrome in which there were pathologic features common to both the latter disease and to Schilder's disease. Considering the rarity of lesions of the spinal cord in Schilder's disease and the comparative frequency of cerebral lesions in neuromyelitis optica, these authors classified their case as one of the latter. Milian and associates [33] stated

31. Michaux reviewed the following reports: (*a*) Holden.[60] (*b*) Holmes.[75] (*c*) Goulden.[10] (*d*) Henneberg.[13d] (*e*) Abelsdorf.[13c] (*f*) Dollfuss, M. A.: Un cas de neuropticomyélite optique à évolution suraiguë, Ann. d'ocul. **163**:115-122, 1926. (*g*) Dechaume, J.: La neuropticomyélite aiguë: maladie à virus neurotrope; recherches expérimentales, Lyons, Edition du Service Photographique de l'Université de Lyon, 1926. (*h*) Euzière, P., and Bremond, R.: À propos d'un cas de neuropticomyélite, Rev. d'oto-neuro-opht. **5**:129-132, 1927. (*i*) Beck.[12] (*j*) Marinesco, G.: Un cas exceptionnel d'ophtalmo-neuro-myélite, Rev. d'oto-neuro-opht. **5**:379-380, 1927. (*k*) Vedel and Puech: Neuromyélite optique aiguë, ibid. **5**:125-128, 1927. (*l*) van Bogaert, L.: Neuro-myélite optique aiguë avec dissociation albuminocytologic du liquide céphalo-rachidien, J. de neurol. et de psychiat. **27**:106-107, 1927. (*m*) van Gehuchten, P., and Gaudissart, P.: Un cas de neuro-myélite optique: La stase papillaire dans les myélites; L'étiologie de la neuro-myélite, Rev. d'oto-neuro-opht. **5**:541-550, 1927. (*n*) van Gehuchten, M. P.: Deux cas de neuro-myélite optique, J. de neurol. et de psychiat. **27**:65-66 and 80, 1927. (*o*) Koelichen, M.: Cas de neuro-myélite optique à issue favorable, Rev. neurol. **2**:372-373, 1928. (*p*) Guillain, G.; Alajouanine, T.; Bertrand, I., and Garçin, R.: Sur une forme anatomo-clinique spéciale de neuro-myélite optique nécrotique aiguë avec crises toniques tétanoïdes: Contribution à l'étude des crises toniques sous-corticales, Ann. méd. **24**:-24-57, 1928.

32. Marinesco, G.; Draganescu, S.; Sager, O., and Grigoresco, D.: Un cas anatomo-clinique d'ophtalmo-neuro-myélite: Sur les relations de cette affection avec la maladie de Schilder et la sclérose en plaques, Rev. Neurol. **54**:193-228, 1930.

33. Milian; Lhermitte; Schaeffer, and Horowitz: La neuropticomyélite aiguë: observation anatomo-clinique, Rev. neurol. **56**:257-279, 1931.

the opinion that neuromyelitis optica is a virus disease and attempted to isolate the virus in their case. They performed corneal scarification tests and intracerebral inoculations in monkeys and dogs with the cerebrospinal fluid. These investigators expected to find an unidentified neurocytotropic virus but were unsuccessful. Van Bogaert presented a case of neuromyelitis optica to the Paris Neurological Society in 1927 [33]; in 1932 he [34] republished the case, after the death and autopsy of the patient, as multiple sclerosis. In van Bogaert's estimation, the original diagnosis was an error, the condition later proving to be multiple sclerosis. Ferraro [35] did not subscribe to van Bogaert's conclusion and offered this case as evidence of the difficulty in differential diagnosis of these two conditions. In 1934 Cestán, Riser and Planques [36] reported a case in which the typical lesions of neuromyelitis optica were associated with demyelination in the brain stem, cerebellum and cerebrum. Uréchia and Veluda [37] reported a case in which death occurred five days after the initial symptoms. In 1935 Alajouanine and associates [38] described lesions microscopically identical with those of multiple sclerosis in a case clinically typical of neuromyelitis optica; they concluded that these syndromes are part of the same disease. Kipfer [39] in 1945 presented a case with complete blindness that responded well to sulfonamide therapy; there were return of light perception in three days, regression of the myelitic symptoms in one week and almost complete cure of both in one year. Admitting the doubtful effect of the medication, Kipfer stated that the regression was so prompt and so spectacular that the case could not represent a coincidence of therapy and spontaneous remission.

German Literature.—In 1932 Kyrieleis [40] reported several cases of neuromyelitis optica; he expressed the belief that this disease repre-

34. van Bogaert, L.: Erreur de diagnostic: neuromyélite optique aiguë: premier stade d'une sclérose en plaque typique, J. de neurol. et de psychiat. **32:** 234-247, 1932.

35. Ferraro, A.: Primary Demyelinating Processes of the Central Nervous System: An Attempt at Unification and Classification, Arch. Neurol. & Psychiat. **37:**1100-1160 (May) 1937.

36. Cestán, Riser and Planques: De la neuro-myélite optiques, Rev. neurol. **62:**741-762, 1934.

37. Uréchia, C. I., and Veluda, P.: Neuromyélite optique aiguë avec controle anatomique, Rev. neurol. **61:**1039-1041, 1934.

38. Alajouanine, T.; Hornet, T.; Thurel, R., and Rossano, R.: Un cas anatomo-clinique de sclérose en plaques aiguë avec symptomatologie de neuropticomyélite, Rev. neurol. **63:**98-109, 1935.

39. Kipfer, M.: Neuromyélite optique d'evolution suraiguë quérie après sulfamido thérapie intensive, Rev. neurol. **77:**151-153, 1945.

40. Kyrielis, W.: Ueber örtlich und zeitlich gehäuftes Auftreten von Neuritis nervi optici und Neuritis retro-bulbaris, sowie ihre Beziehungen zur Encephalomyelitis disseminata und zur multiplen Sklerose, Deutsche Ztschr. f. Nervinh. **124:**220-238, 1932.

sented an abortive form of multiple sclerosis and explained the clinical differences by postulating a variable intensity of the etiologic agent combined with a variable reaction in the patient. Klar [41] described 4 cases of this disease with severe monocular retrobulbar neuritis and total amaurosis, followed in a few days by the same process in the other eye. Klar inoculated the cerebrospinal fluid from his patients into the corneas of rabbits and produced superficial punctate keratitis. Ter Braak and van Herwaarden [42] in 1933 reported the ocular findings in 6 cases of neuromyelitis optica; their ocular findings consisted of (1) perivasculitis, (2) diffuse retinitis and (3) choroiditis. Popow[43] in 1935 reported a case of Devic's disease in which the blood culture was positive for staphylococci and the histologic examination of the spinal cord led to the diagnosis of purulent myelitis. Peters,[44] discussing 2 cases of this syndrome, differentiated neuromyelitis optica and multiple sclerosis on a pathologic basis. In 1936 Markiewicz and Peters [45] again stressed the difference in pathology in their presentation of 2 more cases.

Other Reports.—In the Italian literature, Salvati.[46] reviewed the subject of neuromyelitis optica in 1931; he stated that the disease was an inflammation of the interstitial tissue of the nervous system. Gozzano [47] reviewed the literature again in 1937 and stated that the differences between neuromyelitis optica and multiple sclerosis are very great, although he considered them both virus diseases. In the Spanish literature, case reports have been published by Barcia Goyanes [48] and López Ibor.[49] In Russia, the disease has been reported by Shenderov,[50]

41. Klar, J.: Encephalomyelitis optica acuta, Klin. Monatsbl. f. Augenh. **89**:645-655, 1932.

42. ter Braak, J. W. G., and van Herwaarden, A.: Ophthalmo-Encephalo-Myelitis: encephalomyelitis disseminata mit ungewöhnlichen Augenerscheinungen, Klin. Monatsbl. f. Augenh. **91**:316-343, 1933.

43. Popow, N. A.: Neuromyelitis optica acuta, Deutsche Ztschr. f. Nervenh. **135**:142-157, 1935.

44. Peters, G.: Zur Frage der Beziehungen zwischen der disseminierten, nichteitrigen Encephalomyelitis und der multiplen Sklerose, Ztschr. f. d. ges. Neurol. u. Psychiat. **153**:356-384, 1935.

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48. Barcia Goyanes, J. J.: Un neuvo caso de sindrome de Devic, Crón. méd., Valencia **37**:687-690, 1933.

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50. Shenderov, L. A.: Neuromyelitis After Influenza, Sovet. nevropat., psikhiat., i psikhogig. **1**:639-645, 1932.

Ammasov [51] and Drombravskaya,[52] and in Turkey, by Mazal.[53] The first case of the disease diagnosed in China was described by Chang [54] in 1934. Recently, a case report by Stern [55] appeared in Palestine (1945), and by De and Chatterjee,[56] in India (1946).

NOTE.—Because of the limitations of space, no attempt has been made in this historical note to include all the papers and case reports on neuromyelitis optica. The number of publications on this rare disease is surprising. A supplementary list of references is appended to this paper, including many references prior to 1927 that were not listed in previous bibliographies.

REPORT OF CASES

CASE 1.—Mrs. S. S., a Negro woman aged 31, was admitted to the Neurological Institute on Jan. 18, 1936 because of numbness and weakness of the extremities and blurred vision in the left eye. The patient first noted the blurring of vision three weeks prior to admission. Four days before admission she noticed that her right thumb and the radial aspect of her right arm were "going to sleep." A few hours after this began, the entire right arm felt numb and weak; there was no pain. On the following day the numbness descended to the right leg, and by evening the entire right half of the body was numb and weak. Then the numbness appeared in the left foot and ascended to the knee. On the day of admission the patient observed the same symptoms in the fingers and palm of the left hand.

Past History.—The patient had the usual diseases of childhood. She had influenza in 1918, otitis media in 1928 and pneumonia in 1927. She had suffered from sinus trouble for three years. She had one child who was living and well; there had been but the one pregnancy. There was no history of familial disease. She was employed as a domestic. The remainder of the history was noncontributory.

Physical Examination.—The patient, a well developed young woman, exhibited difficulty in controlling the movements of her arms and hands, a bilateral wrist drop and an unsteady gait. The temperature, pulse, respiration and blood pressure were normal. Reflexes were hypoactive in the arms and hyperactive in the legs. A Babinski sign was elicited bilaterally, and abdominal reflexes were absent on the left. The pupillary reaction to light was sluggish, and there was questionable pallor of the left optic disk. Sensory examination showed hypesthesia over both upper and lower extremities. Position sense was impaired in the arms, and vibratory sense was lost in the legs. Pain and temperature sensations were diminished on the right side, with patchy involvement on the left. Physical examination revealed nothing else abnormal except for kyphoscoliosis.

51. Ammasov, M. M.: Clinical Aspects and Pathologic Anatomy of Neuromyelitis Optica, Sovet. psikhonevrol., no. 3, 5-14, 1936.

52. Drombravskaya, R. I.: Optic Encephalitis, Klin. med. **19**:50-55, 1941.

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54. Chang, S. P.: Acute Retrobulbar Neuritis in Myelitis: Report of a Case, Chinese M. J. **48**:991-998, 1934.

55. Stern, A.: Acute Multiple Sclerosis with Symptomatology of Optic Neuromyelitis, Harefuah **29**:236-237, 1945.

56. De, M. N., and Chatterjee, J. R.: A Case of Neuromyelitis Optica, Indian M. Gaz. **81**:361-362, 1946.

Ophthalmologic Consultation (Jan. 25, 1936).—Vision was 20/20 in the right eye and 20/200 in the left eye. Externally, the eyes were normal. The anterior chamber and media were clear in both eyes. The pupillary reflexes in the right eye were normal; the pupil in the left eye was sluggish, and its excursion was only half that of the right eye. The fundus was normal in the right eye, but the optic disk on the left showed primary optic nerve atrophy. The peripheral fields were within normal limits. The impression was stated that there is nothing in the eyegrounds to indicate an acute process; on the contrary, the fundus on the left appeared to be an old process.

January 27: Another ophthalmologist saw the patient and found a large central scotoma in the left eye. He described the left optic disk as showing moderate temporal pallor. The impression was that of "recent retrobulbar neuritis of the left eye, most likely caused by whatever had produced the other neurologic changes."

March 4: The central fields showed a large centrocecal scotoma on the left (fig. 1); the peripheral fields were normal.

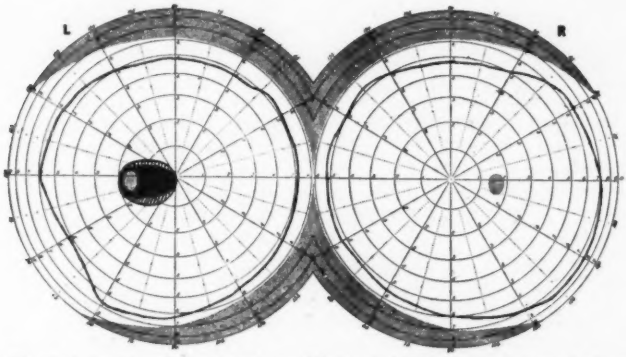

Fig. 1 (case 1).—Central fields on March 4, 1936. The cross patched areas show scotoma for 1 mm. red test object; the black area, scotoma for 2 mm. white test object.

Laboratory Findings.—The blood count was normal until near the patient's death, when the white cell count was 13,000, with a normal differential distribution. The Wassermann reaction was negative on all occasions. Repeated examinations of the urine revealed few to many white cells, an acid reaction, normal specific gravity and no sugar or acetone. Examination of the spinal fluid on four occasions, with approximately the same results, disclosed a clear fluid; normal dynamics; 10 cells per cubic millimeter; a 1 plus reaction for globulin; 56 mg. of protein, 62 mg. of sugar and 702 mg. of chlorides, per hundred cubic centimeters, a negative Wassermann reaction and a colloidal gold curve of 1233332100 on two occasions. All other laboratory findings were normal.

Course.—It was the impression of the staff that the patient had an acute intrinsic disease of the central nervous system, either neuromyelitis optica or neurosyphilis. Provocative Wassermann tests were done, with negative reactions of both the blood and the spinal fluid. The patient's symptoms and neurologic status changed from day to day, and the findings varied with different examiners.

On January 30, urinary retention developed, necessitating catheterization. Her urinary difficulty cleared up, only to recur in a few weeks. Spinal drainage was performed, without benefit. Paralysis of the extremities was progressive and became severe in all four limbs. The sensory disturbances progressed until all sensory functions were abnormal to the third cervical segment. She complained of excessive perspiration about the head and neck and had episodes of hiccups. Motor function returned temporarily to a considerable degree on the right side. On the other hand, there developed profound motor palsies in the left arm and leg, which were severer than were ever noted on the right. No spasticity was seen; instead, hypotonicity was the rule. The patient became stuporous the day before her death. Respiration became rapid and shallow; then just before death, extremely slow, and finally imperceptible. She died March 9, approximately two and a half months after the onset of her visual difficulty.

Clinical Diagnosis.—The clinical diagnosis was encephalomyelitis.

Autopsy.[57]—Macroscopic Observations: External examination of the body revealed nothing remarkable. Internally, the body cavities and the enclosed viscera appeared normal. The brain, cerebellum, brain stem and spinal cord appeared (autopsy of the nervous system) grossly normal. On section, the cervical portions of the spinal cord revealed indistinct margins between the white and the gray matter and a yellowish gray discoloration of the posterior columns and the left lateral column. There were similar lesions in the upper half of the thoracic portion of the cord.

Microscopic Observations: The abdominal and thoracic organs all appeared normal except the lungs, which showed a typical picture of active bronchopneumonia.

Brain and brain stem. The cerebrum showed congestion and edema and some diffuse loss of ganglion cells in the cortex, but no other changes of note. The brain stem was congested and contained occasional small perivascular hemorrhages. The pia of the medulla was infiltrated with lymphocytes and plasma cells, and the parenchyma of the medulla was congested. The Marchi stain of the lower part of the medulla showed scattered foci of degeneration, and the Weigert stain revealed occasional small foci of demyelination. These areas were edematous, and the ganglion cells were pale and partially degenerated. There was some microglial proliferation. The pons showed occasional perivascular infiltrations and hemorrhages.

Optic nerves. Hematoxylin and eosin stains of the left optic nerve revealed homogeneous, pale pink interstitial tissue between the nerve bundles, with little of the normal architecture remaining. These spaces were rich in microglia cells and hypertrophied astrocytes; the microglia cells were seen in all stages of transition, from rod cells to gitter cells. The microglia cells were most numerous within the degenerated areas, while the astrocytes were commoner at the margins of these areas. There was extensive perivascular infiltration of lymphocytes and plasma cells, both in the nerve substance and in its meninges. With phosphotungstic acid stains, the astrocytes were especially well demonstrated. The Spielmeyer and Weigert stains showed these areas to be extensively demyelinated. The Bodian stain demonstrated a decrease in the number of axons and severe pathologic changes in the remaining fibers, which were poorly stained, fragmented and very irregular in caliber. The presence of neutral fat shown with the Marchi stain demonstrated that the process was comparatively recent, and probably still active. There was no evidence of hyperplasia of glial fibers.

57. Autopsies of the nervous system in this case and in the other cases reported were performed by Dr. Abner Wolfe.

The right optic nerve showed changes similar to those in the left nerve, but of considerably milder degree. (There was no clinical evidence of involvement of this nerve.) Figure 2 is a photograph of a Weigert stain of the two nerves. The optic chiasm showed the same diffuse demyelination, which was again severer

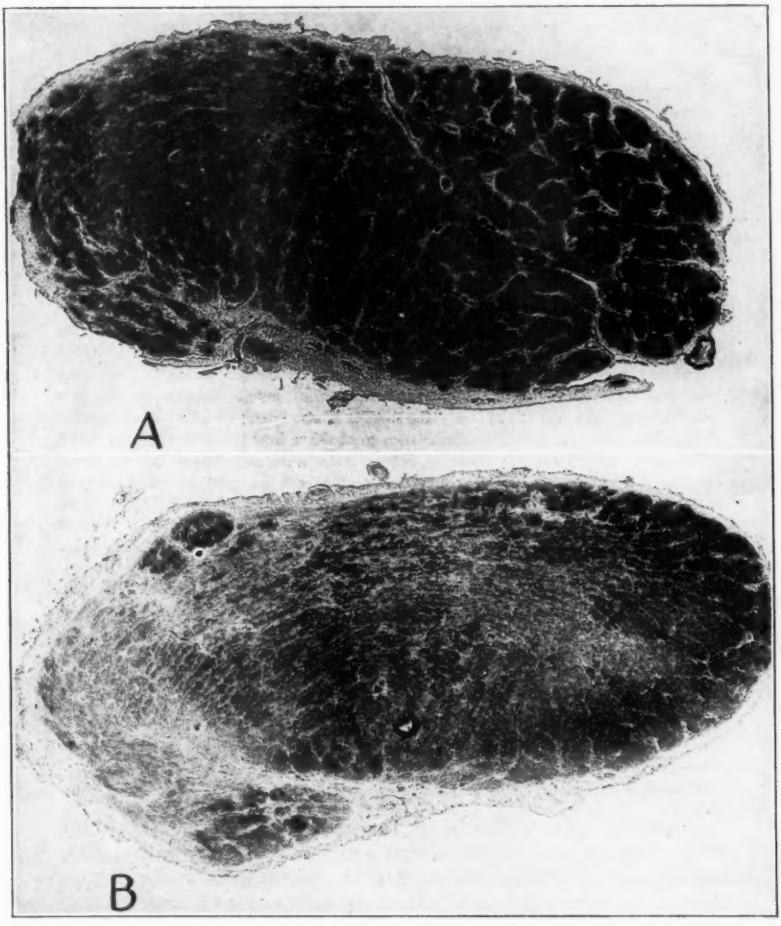

Fig. 2 (case 1).—Weigert stains of the optic nerves. (*A*) The right optic nerve appears grossly almost normal but microscopically shows slight diffuse demyelination. (*B*) The left nerve shows extensive demyelination of the entire cross section, although severer in some areas than others.

on the left side. The fibers from the left optic nerve which enter into the decussation are seen to be almost entirely devoid of myelin sheaths.

Spinal cord. The hematoxylin and eosin stains show diffuse infiltration of the leptomeninges with lymphocytes and plasma cells. A similar infiltration was seen about the blood vessels entering the parenchyma of the cord from the surface. In the cervical sections, the greater part of all the white columns was undergoing intense degeneration. In the thoracic segments, there was advanced degeneration of the periphery of the posterior and lateral white columns and parts of the anterior columns. In addition to these more extensive lesions, there were many minute foci throughout the remaining white matter and pronounced necrosis of the central gray substance.

Histologically, these areas proved to be foci of softening or necrosis, with loss of axons, myelin sheaths and neuroglia. Within these areas there were many compound granule cells, as well as a multiplication of microglia cells, many of which were in the process of conversion to compound granule cells. In general, the outer edge of the necrotic band was broken down and edematous, while its inner aspect showed greater infiltration and progression of the process. Partially degenerated zones were seen, containing bright red fragments of swollen axons, degenerating astrocytes, proliferating microglia cells and developnig compound granule cells. Occasional corpora amylacea were seen developing from degenerated astrocytes. In the more severely affected foci, fragmentation of fibers and clasmatodendrosis of the astrocytes were seen. The phosphotungstic acid hematoxylin stain revealed that many of the astrocytes near the margins of the degenerated zones were hypertrophied; within the zones they were partially or completely necrotic. The Spielmeyer stain revealed demyelination of the areas just described, with swelling and fragmentation of myelin sheaths in the less severely affected areas. Frequently the smaller areas of demyelination were observed to be perivascular, and these could be seen to coalesce into the larger lesions. With the Marchi stain, irregular areas of degeneration were observed in all the white columns. There were intense congestion and edema, as well as some necrosis in the anterior and posterior gray horns. Many of the nerve cells of the anterior horn were undergoing degeneration; these were deformed, lacking in their Nissl substance, pale staining, filled with an excess of lipid and occasionally vacuolated. There was some multiplication of microglia cells here as well. There were also foci of demyelination in the anterior and posterior roots.

The lumbar and sacral segments were only minimally involved. The dorsal ganglions showed mild infiltration of lymphocytes and plasma cells. Some of the ganglion cells were vacuolated and laden with lipid. With the phosphotungstic acid hematoxylin stain, some of the axons in the roots appeared irregularly swollen. With the Weigert stain sections of the right sciatic nerve showed occasional degenerated myelin sheaths. There was Marchi degeneration of scattered sheaths in each bundle of this nerve. With the Weigert stain the left brachial nerve showed even more degeneration than did the sciatic nerve.

Anatomic Diagnosis.—The anatomic diagnosis was neuromyelitis optica.

Summary.—The patient, a young Negro woman, had blurred vision of the left eye and numbness and weakness in the arms and legs. Examination on her admission revealed an unsteady gait, ataxia, hyporeflexia in the arms and hyperreflexia in the legs, with a bilateral Babinski sign. She had mild pallor of the left optic disk and a central scotoma. Sensation was impaired throughout. The spinal fluid was normal except for the presence of a small amount of globulin. There was evidence of disseminated lesions in the central nervous system, and the diagnosis

rested between neuromyelitis optica and neurosyphilis. The course was progressive, although the symptoms varied from day to day. The patient finally became stuporous and died two and a half months after the onset of her visual difficulty.

Autopsy revealed diffuse demyelination of both optic nerves, although there was no clinical evidence of damage to the right nerve. In the spinal cord, advanced necrosis was present throughout the thoracic and cervical levels. Intense demyelination was noted in the white columns; the demyelination was irregular, not involving any definite systems. The lesions did not appear to be essentially perivascular, although they might have been the result of coalescence of perivascular foci. There were intense multiplication of the microglia cells and conversion into fat granule cells. There was extensive perivascular infiltration of lymphocytes, plasma cells and monocytes. Pathologically, this case must be considered one of disseminated encephalomyelitis, of the type known as "optic neuromyelitis."

Case 2 (presented with the permission of Dr. Gordon M. Bruce).—W. E., a white man aged 57, a carpenter, was referred to Dr. Gordon M. Bruce, on July 1, 1938, with a history of blurred vision for three weeks. The patient had had good health and good vision until March 1938, when he began to have pains behind the eyes. After a change in glasses by his local oculist the pains gradually ceased. Early in June he noticed blurring of vision of the left eye and, after a few days, in the right eye. On June 20 he became conscious of numbness of the face, which he attributed to the extraction of teeth. The patient's family history was not unusual, and his past history revealed only the usual diseases of childhood, a history of hay fever for twenty-five years and allergy to bananas and cheese.

Ophthalmologic Examination (July 1).—Visual acuity was 20/40 in the right eye, and 1/200 in the left eye: there was no improvement with glasses. Externally, the eyes appeared normal. The pupils were round, equal and active. Tension was normal in both eyes. Ophthalmoscopic study revealed a small area of old chorioretinitis in the periphery of the right eye; otherwise the fundi were normal. The peripheral fields were normal. The tangent screen revealed enlargement of the blindspot on the right and a very large central scotoma, involving the blindspot, on the left (fig. 3*A*). Roentgenograms of the orbits were normal. A diagnosis of acute retrobulbar neuritis was made, and the patient was admitted to the Institute of Ophthalmology for study.

Physical Examination.—The patient, an elderly white man, did not seem acutely ill. The temperature, pulse, respiration and blood pressure were normal. He possessed many carious teeth. His heart was slightly enlarged to the left, but physical examination was otherwise noncontributory. Neurologic examination gave normal findings except for a positive reaction to the Romberg test.

Laboratory Findings.—The blood count showed 16,700 white cells, with 89 per cent polymorphonuclear leukocytes, 9 per cent lymphocytes and 2 per cent monocytes. The sedimentation rate was 30 mm. per hour. The urine was normal, and the Wassermann reaction of the blood was negative. Roentgenograms of the chest and skull were normal. Examination of the spinal fluid revealed clear fluid; normal dynamics; no cells; a positive reaction of the Pandy test; 59.5 mg. of protein, 95 mg. of sugar and 681 mg. of chlorides, per hundred cubic cen-

timeters; a colloidal gold curve of 1111100000, and a negative Wassermann reaction. All other laboratory findings were within normal limits.

Course.—With hospitalization, vision improved in the left eye; at the same time, it decreased rapidly in the right eye. The central fields on July 15 showed shrinkage of the central defect on the left and a new large centrocecal scotoma on the right (fig. 3*B*). Vision was not tested at this time. Neurologic consultation disclosed staggering with a wide base; a positive Romberg sign; poor

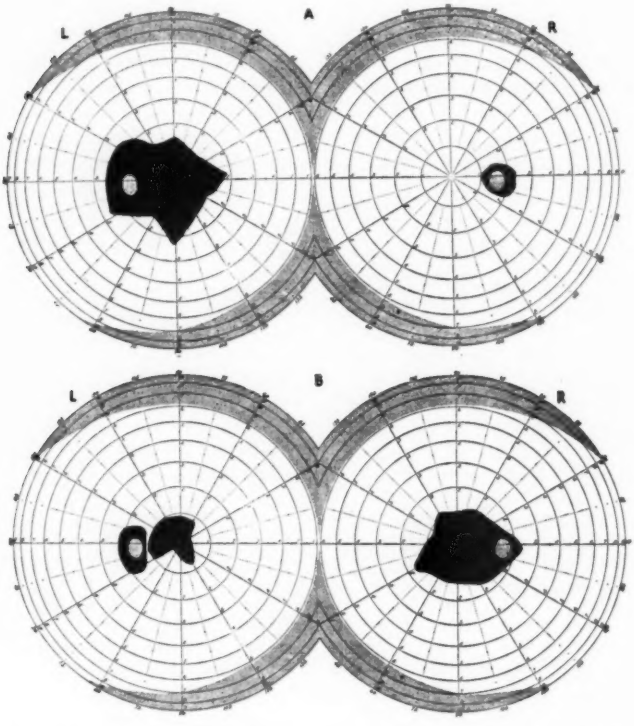

Fig. 3 (case 2).—*A*, central fields taken on July 1, 1938, showing enlargement of the blindspot of the right eye and a large centrocecal scotoma on the left (3 mm. white). Corrected vision was 20/40 in the right eye and 1/200 in the left eye. *B*, central fields on July 15, showing shrinkage of the central defect of the left eye, and a new large centrocecal scotoma on the right (3 mm. white). These fields are reproduced by courtesy of Dr. Gordon M. Bruce.

coordination; hyperreflexia, more pronounced on the right side; weakness of all four extremities; and normal sensation. The neurologic consultant discarded multiple sclerosis as a diagnosis because of the patient's age and suggested an allergic process, causing focal edema in the optic nerves and cerebellar pathways.

The patient was placed on a vigorous dehydration program but failed to improve. His neurologic symptoms gradually became more pronounced, and he fell several times when trying to walk alone. On July 20 he was transferred to the Neurological Institute, unimproved.

On July 21 the patient complained of a tingling sensation in his arms and legs; this was most pronounced in the hands and feet, extending up the arms to the elbows and up the legs to the knees. He also noticed a noise in his ears, not associated with any diminution in hearing. The next day he complained of great dizziness. Examination on July 26 revealed increased staggering; marked ataxia; increased muscular weakness of the extremities; hyperreflexia; questionable blurring of the optic disks; irregular jerky nystagmus on lateral gaze; reduced pharyngeal reflex, and a deviation of the uvula to the left. Vision was then 20/200 in each eye. His condition grew rapidly worse, and he began to exhibit signs of respiratory embarrassment. He died on July 31, with respiratory failure. The

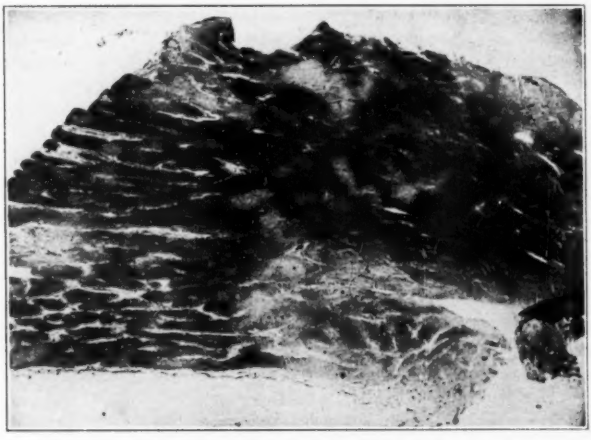

Fig. 4 (case 2).—Smith-Quigley stain of the right optic nerve, showing a number of small foci of demyelination and one large coalescent focus below.

patient showed such a multiplicity of neurologic symptoms and signs that it was impossible to explain them on the basis of any single lesion in the central nervous system.

Clinical Diagnosis.—The clinical diagnosis was encephalomyelitis.

Autopsy.—Macroscopic Observations: Externally, there was only mild wasting of the muscles of the arms and hands. (Permission limited the examination to the brain and spinal cord.) The brain, cerebellum, brain stem and spinal cord appeared normal externally and on cut section.

Microscopic Observations: Brain and brain stem. The leptomeninges were congested and contained occasional petechial hemorrhages and scattered collections of lymphocytes. Many of the nerve cells of the cerebrum were pale staining and showed chromatolysis. The Pal-Weigert stain revealed no demyelination of these tissues.

Optic nerves. With the hematoxylin and eosin stain, the optic nerves showed coalescent areas of necrosis with infiltration by polymorphonuclear leukocytes, lymphocytes and monocytes. In these areas there were edema, destruction of the neural elements, conversion of microglia into fat granule bodies, degeneration of astrocytes and oligodendrocytes and proliferation of capillaries. Lipid-laden phagocytes were packed in the perivascular spaces and lymphocytic masses were seen around the blood vessels. In the more severely involved foci, there were a loss of architectural markings and more abundant perivascular exudate. The Smith-Quigley stain showed small discrete and large coalescent areas of demyelination (fig. 4); in these necrotic foci a few poorly preserved myelin sheaths were still present. With the phosphotungstic acid hematoxylin stain, sections of both nerves showed pronounced astrocytosis.

Pons. The meninges were infiltrated and congested, as already noted. Many of the vessels penetrating the parenchyma from the meninges showed similar perivascular infiltration of lymphocytes and plasma cells. A number of large necrotic lesions affected most of the structures of the reticular portion of the pons and a single large lesion involved the center of one-half the tegmentum. These lesions showed edema, necrosis and degeneration of the tissues, similar to the lesions in the optic nerves. There was conversion of microglia into lipid-scavenging phagocytes throughout the sections. There were fragmentation and destruction of the interstitial tissue. The nerve cells in the nuclei outside the lesions contained excessive lipid material, and some were partially destroyed. The Pal-Weigert stain demonstrated intense demyelination of the lesions previously described (fig. 5), although small numbers of preserved myelin sheaths were seen to pass through the softened areas. At the margins of these areas myelin sheaths were undergoing fragmentation and degeneration, showing that the lesions were still active. Some of the demyelinated areas were sharply demarcated, particularly the one in the tegmentum, but many of those in the reticular area were confluent. The Cajal stain showed small numbers of hypertrophied astrocytes about the margins of these lesions.

Spinal cord. The meninges and parenchyma were congested, and many of the anterior horn cells showed excessive lipid material. There were no other changes of note.

Anatomic Diagnosis.—The anatomic diagnosis was neuromyelitis optica.

Summary.—A middle-aged man suffered sudden retrobulbar pain for a few days, followed in three months by blurring of vision. He began to have difficulty in walking, staggering but not falling. There then developed a varied assortment of motor, sensory and cranial nerve symptoms and signs, and the patient eventually died of respiratory failure, four months after the onset of visual difficulty.

Autopsy revealed acute necrotic perivascular lesions in the pons and optic nerves. Microscopically, the main features were demyelination, destruction of axons, proliferation of astrocytes, proliferation and conversion of microglia into fat-scavenging phagocytes and perivascular infiltration of lymphocytes, monocytes and plasma cells.

The pathologic and clinical pictures are those of neuromyelitis optica, but the process was observed only in the optic nerves and pons. The absence of lesions in the spinal cord was unusual. In view of the fact

that the lesions in the spinal cord in neuromyelitis optica may be confined to a few segments, one might consider this an analogous state, in which the pathologic lesions were confined to the brain stem instead.

Case 3.—Miss L. J., an 18 year old Jewish girl, had onset of weakness of the left arm and leg on April 1, 1941. On April 10 she noticed clumsiness of the left hand and numbness of the left side of the body. These disabilities were

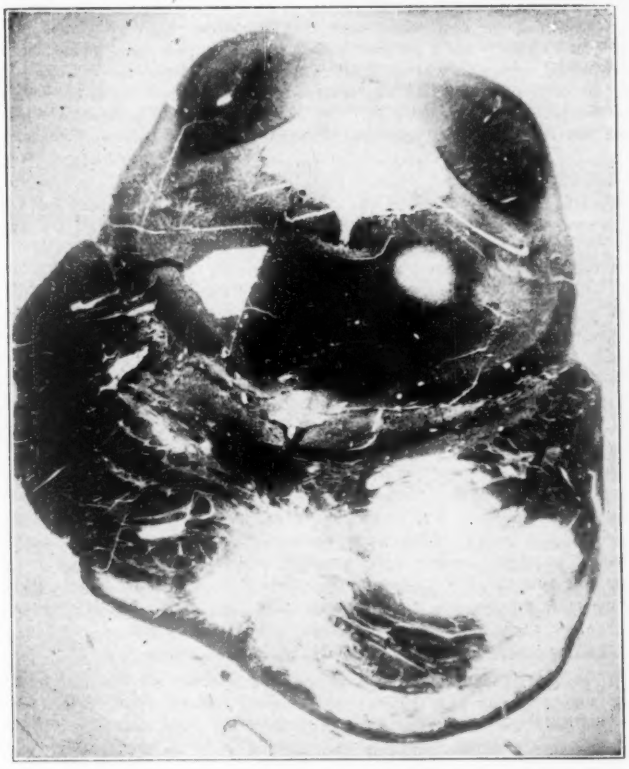

Fig. 5 (case 2).—Pal-Weigert stain of the pons, showing extensive areas of severe demyelination.

progressive, and her gait became unsteady. Blurring of vision was first noted on April 20. She became euphoric and confused. On April 24 she was admitted to the Neurological Institute for study.

Physical Examination.—The patient, a well developed girl, appeared drowsy and somnolent. The temperature, pulse, respiration and blood pressure were normal. Neurologic examination revealed weakness of the legs, with inability

to stand alone; slight pallor of the optic disks; equal and symmetric pupils, which reacted to light and in accommodation; poor convergence; defective conjugate movements of the eyes, anesthesia of the trigeminal nerve on the left; weakness of the left side of the face; protrusion of the tongue to the right; poor coordination; slurring of speech; a positive Babinski sign on the right, and normal sensation over the body. The remainder of the physical examination showed nothing abnormal.

Laboratory Findings.—The blood count showed leukocytosis, with a count of 16,450 with 89 per cent polymorphonuclear leukocytes, 9 per cent lymphocytes, 1 per cent monocytes and 1 per cent eosinophils. The urine was normal. Examination of the spinal fluid revealed clear fluid; normal dynamics; 9 white blood cells per cubic millimeter; 68 mg. of protein and 65 mg. of sugar per hundred cubic centimeters; a colloidal gold curve of 1100000000, and a negative Wassermann reaction. All other laboratory findings were within normal limits.

Course.—On April 26 weakness of the right arm and leg developed. She was confused, forgetful and euphoric most of the time. The Babinski reflex was elicited bilaterally, and sensation was lost over the left side of the body below the fourth thoracic segment. On April 30 she was disoriented as to time and place. The pupils were large, equal and symmetric and reacted poorly to light. She could not converge her eyes, and there was paralysis of upward gaze. Sensory loss over the right side of the face developed. Deep reflexes were hyperactive in the right arm and the left leg, and the abdominal reflexes were lost. Poor vision developed bilaterally (it was normal before admission, according to her family physician), but study of the fundus revealed no abnormality. (Visual acuity was not determined.) One examiner noted normal excursions of the right eye, with limitation of motility of the left eye in all directions; he also noted left homonymous hemianopsia. On May 1 the patient lapsed into coma and became incontinent of feces and urine. On May 3 the pupils were widely dilated, with no reaction to light or convergence; there were paralyses of the third and sixth nerves bilaterally. At this time she showed left hemiplegia, with a spastic arm and a flaccid leg; there was right hemiparesis, severer in the leg. On May 4 the patient had complete bilateral ophthalmoplegia, and the fundi still appeared normal. She slowly became more somnolent and exhibited a tendency toward decerebrate posture. Her temperature began to rise; respiration became slower, and the pulse became slow and indistinct. She died on May 13 of respiratory paralysis, six weeks after the onset of motor and sensory symptoms.

Clinical Diagnosis.—The diagnosis was encephalomyelitis.

Autopsy.—Macroscopic Observation: External examination was noncontributory. Internally, the body cavities and enclosed viscera were within normal limits. Grossly, the brain, cerebellum, brain stem and spinal cord appeared normal. On section of the cerebrum, the centrum ovale on each side contained numbers of sharply demarcated lesions, which were salmon colored and studded with yellow punctate areas. Section of the brain stem revealed a pyriform lesion in the right cerebral peduncle. The pons also contained several small lesions. The upper cervical segments of the spinal cord showed a wedge-shaped lesion in the right lateral white column. There were scattered small lesions through the rest of the cervical portion of cord and in the sections of the lumbar portion of the cord, but no lesions were seen in the thoracic segments.

Microscopic Observations: The sections of the thoracic and abdominal viscera were within normal limits.

Cerebral cortex. There were many lesions in the subcortical white matter; the smallest of these were perivascular. Often, however, these perivascular lesions coalesced into larger lesions, so that the appearance was that of an irregular patch of softened white matter, which was edematous and diffusely infiltrated with lipid-laden phagocytes and small numbers of lymphocytes. There was degeneration of all the neural elements in these patches, with destruction of myelin sheaths, axons and neuroglia. The Mahon stain demonstrated nearly complete loss of myelin sheaths within these lesions, although fragments of myelin of varying size were seen at the margins of the foci. The Bodian stain disclosed the preservation of some axons, even in the areas with the greatest necrosis; these remaining axis-cylinders occasionally showed swelling and fragmentation. With the Nissl stain, occasional cells showed loss of Nissl substance. Moderate astrocytosis was revealed by the phosphotungstic acid hematoxylin stain but appeared more prominent than it actually was because of the size attained by these hypertrophied astrocytes.

Centrum ovale: Lesions similar to those just described were also noted in this area. The Mahon stain showed extensive demyelination, which was perivascular, although some large, coalesced lesions could be seen.

Midbrain. The substantia nigra and the cerebral peduncle on one side contained a large, coalesced lesion. There were another lesion just lateral to the aqeduct of Sylvius and a few small scattered lesions. The Mahon stain showed total demyelination in these foci. With the Bodian stain, the discrepancy between the loss of axons and the myelin sheaths is more striking here than in any of the preceding sections.

Pons. Large, irregular lesions were present in the reticular zone, and a number of small foci were seen in the tegmentum. There was intense demyelination in all these lesions. The pictures with the Bodian, phosphotungstic acid hematoxylin and Nissl stains were similar to those already described.

Medulla. Small lesions, similar to those in the pons, were scattered throughout the medulla.

Spinal cord. At the cervical level, there was a large, coalescent lesion in the lateral and anterior white columns, surrounding and involving a portion of one anterior gray horn (fig. 6). In addition, there were numerous small lesions in the marginal white matter. A large, wedge-shaped lesion, similar to that seen in the cervical segments, was also noted in the thoracic sections. At the lumbar level large lesions were present in all but the posterior white columns; occasional small lesions were also seen. Both the anterior and the posterior gray horn were involved in large lesions. The microscopic changes were essentially similar to those described in the cerebrum; some of the nerve cells in the anterior gray horns were, unexpectedly, well preserved.

Optic nerves. There were a number of areas of perivascular infiltration in the connective tissue trabeculae of the right optic nerve associated with microglial proliferation within the nerve bundles. The Mahon stain showed perivascular demyelination in these foci (fig. 7); myelin fragments were still present within the lesions. With the Bodian stain the axons appeared separated and decreased in number within the lesions; none of them appeared swollen or fragmented. A mild astrocytosis was apparent with the phosphotungstic acid hematoxylin stain. The left optic nerve appeared normal.

Anatomic Diagnosis.—The diagnosis was neuromyelitis optica.

Summary.—An 18 year old girl had onset of weakness and numbness of the left side of the body, followed by unsteadiness of gait and

blurring of vision. She became euphoric and confused and then somnolent. The paresis became paralysis and attacked the right side, as did the sensory loss. Her vision became poor, and complete ophthalmoplegia developed. Finally, after six weeks, she became comatose and incontinent and died. Autopsy revealed disseminated foci of necrosis and demyelination in the cerebrum, brain stem, spinal cord and optic nerves. There was destruction of all neural elements, with abundant microglial phagocytosis and moderate astrocytosis. There was considerably more involvement of the brain and the brain stem in this case than in the typical case of neuromyelitis optica.

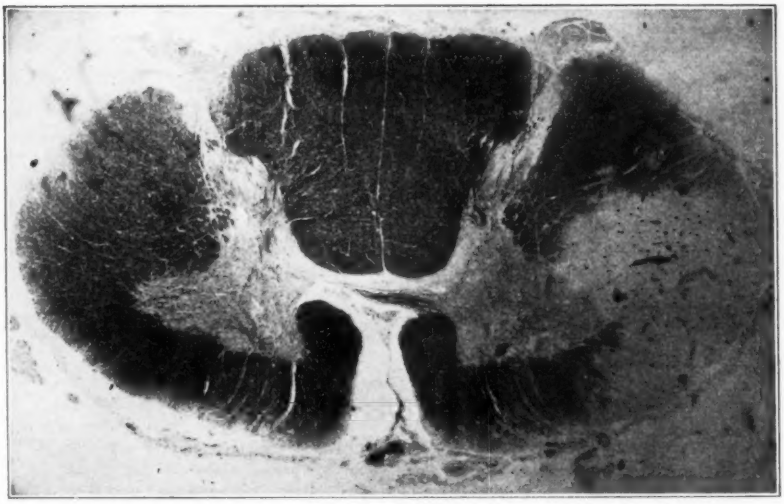

Fig. 6 (case 3).—Mahon stain of a section of the cervical portion of the spinal cord, showing a large lesion involving the lateral and anterior white columns on one side and impinging on the anterior gray horn adjacent.

Case 4.—M. W., a white housewife aged 29, was admitted to the Neurological Institute on Sept. 29, 1945, with the complaints of (1) paralysis of the left leg and weakness of the right leg, (2) numbness on both sides up to the nipple line, (3) urinary and fecal retention and (4) pain in both shoulder girdles. About a week before admission, the patient noticed burning sensations over the abdomen and legs, with sensitivity to touch in the affected areas. She also noted intermittent jerking of the left foot; this involuntary motion continued for about two minutes at a time. She then became aware of numbness in the left foot, which soon appeared in the right foot and progressed up both legs, so that in three days the numbness extended to her breasts. Her legs became so weak that she could not walk, and then she could not move her left leg at all. Urinary retention and constipation developed. Two days before admission pains developed

in her shoulders, neck and back. Tingling, numbness and a cold sensation then appeared in both arms and was beginning to involve the hands at the time of her entrance to the hospital.

Past History.—Two years prior to admission the patient suddenly became blind in the right eye, during an episode of abscessed teeth. The teeth were removed, and two months later her vision return to normal. In October 1944 she was hospitalized for pain in the back and abdomen, associated with constipation. A diagnosis of an acute abdominal condition was made, and an exploratory laparotomy was performed. The appendix and a cyst of the left ovary were removed, and the uterus was found to be enlarged to the size characteristic of a ten weeks' gestation. After operation the patient experienced progressive weakness of both legs, with areflexia and normal sensation. The spinal fluid appeared

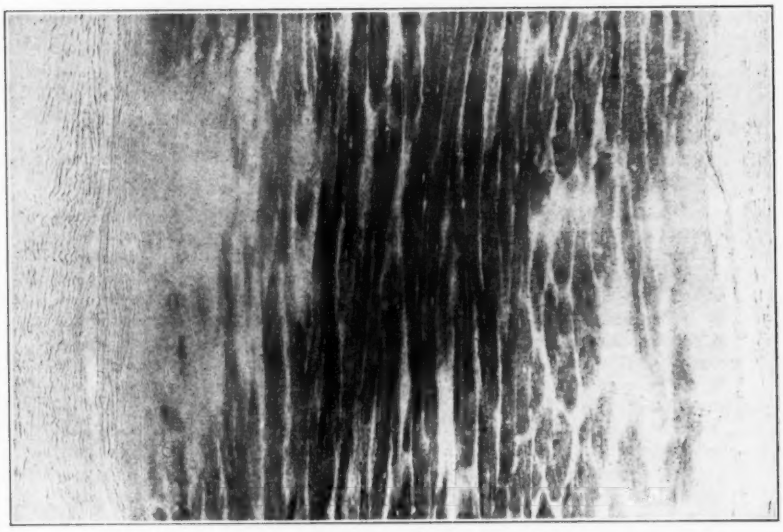

Fig. 7 (case 3).—Mahon stain of a section of the right optic nerve, showing a number of foci of demyelination.

normal on examination. A diagnosis of poliomyelitis was made, and she received the Sister Kenny treatment. In January 1945, with physical therapy, she began to show some improvement in muscular power. In April 1945 she was delivered of a normal child, and in May she was able to walk with crutches. She was discharged from the hospital, with residual weakness of the muscles of the abdomen, back and left thigh.

Physical Examination.—The patient appeared ill and anxious. The temperature, pulse, respiration and blood pressure were normal. Neurologic examination revealed flaccidity of the left leg, with partial paralysis; diminished muscular strength in the right leg; hyperactive tendon reflexes in the arms; areflexia in the legs, with a Babinski sign present bilaterally, and a sensory level at the

fourth thoracic segment. The cranial nerves were intact except for the optic nerves. The remainder of the general physical examination was not significant.

Ophthalmologic Examination.—The left eye was normal. Ophthalmoscopic study of the right eye revealed primary optic nerve atrophy. The pupils were equal and symmetric, and the reactions to light and in accommodation were present but sluggish. The motility of the eyes was normal. Vision was not determined.

Laboratory Findings.—The blood count was normal. Urinalysis showed a moderate number of white cells and a trace of albumin. Examination of the spinal fluid disclosed 86 cells per cubic millimeter, with 99 per cent lymphocytes and 1 per cent polymorphonuclear leukocytes; 240 mg. of protein, 40 mg. of sugar and 701 mg. of chlorides, per hundred cubic centimeters; a normal colloidal gold curve; a negative Wassermann reaction, and sterile cultures. All other laboratory findings were normal.

Course.—During hospitalization the patient had a low grade fever, with temperature usually under 101 F. As motor and sensory symptoms steadily progressed in severity, they also ascended to higher levels, so that on November 4 she began to experience respiratory difficulty. This increased so rapidly that, in spite of sedation and the use of the respirator, she died on November 7, forty-six days after the onset of the acute exacerbation of her illness.

Clinical Diagnosis.—The diagnosis was acute ascending myelitis.

Autopsy.—Macroscopic Observations: External examination revealed severe wasting of the muscles of the extremities. Internally, the heart and the abdominal viscera appeared normal; the lungs showed evidence of lobular pneumonia. The brain and the brain stem appeared normal. The optic nerves and the optic chiasm were flattened, and the right nerve was approximately one half as large as the left. The spinal cord appeared injured in the cervical segments on the left side, and white material oozed from the cut section. The cervical enlargement was swollen, soft and irregular; this swelling extended down to the fifth thoracic level. On section the cord showed pronounced edema of the parenchyma and obscuration of the architectural markings. The central gray matter had an abnormal granular appearance. Little bands of yellowish white material were scattered throughout the section, and in some places the cord was very soft, almost fluid.

Microscopic Observations: Histologically, the sections of the heart, aorta and most of the abdominal viscera were normal. The sections of lung showed active bronchopneumonia, and the sections of liver exhibited advanced atrophy of the parenchyma.

Brain and brain stem. There were no lesions of consequence at this level except for one small perivascular focus of demyelination in the subcortical white matter and occasional perivascular lymphocytic infiltration and edema in the lower portion of the medulla.

Spinal cord. In the cervical sections a large portion of all the white columns were involved in the pathologic process; the anterior and posterior gray horns had also been attacked. In the upper thoracic segments there was a large central area of demyelination, which included most of the gray substance and surrounding white columns; the involved area appeared microcystic. The lower thoracic sections showed widespread extensive demyelination of almost all the white and gray substance. The lumbar sections showed scattered foci throughout.

Histologically, these lesions of the cord were rarefied, microcystic and loose meshed and had lost their normal architectural markings. The process was one of acute necrosis, with destruction of all the neural elements. There were proliferation of microglia and conversion of microglia into phagocytes. There were

hyperplasia of astrocytes in some areas and degeneration of the same cells in others. Where the process had invaded the gray matter, there were destruction of the large nerve cells and the same proliferation of microglia. The perivascular spaces were packed wtih lipid-laden phagocytes. There were rarefied areas containing no cellular structure at all; these appeared to represent old, quiescent foci. The Mahon stain revealed extensive demyelination of all involved areas, although the demyelinated foci varied somewhat in size and in extent of demyelination. About the periphery of some of the lesions, numbers of huge, swollen myelin sheaths were seen undergoing fragmentation. With the Bodian stain, a great loss of axis-cylinders was demonstrated; some of the remaining axons were swollen, distorted and degenerated.

Optic nerves. There was a large area of demyelination in the right optic nerve which involved almost two thirds of the cross section. There were no swollen sheaths or axons in this lesion; apparently, the focus was not active. (These observations corroborated the clinical history.) There was some lymphocytic infiltration of the nerve sheath. With hematoxylin and eosin stain, a mild astrocytosis was seen in the substance of the nerve. The Bodian stain revealed a considerable loss of axis-cylinders in the large lesion. The left optic nerve and the optic chiasm showed no pathologic change in the sections available.

Anatomic Diagnosis.—The diagnosis was optic neuromyelitis.

Summary.—For two years this woman suffered from a subacute relapsing illness in which her eyes and spinal cord were the focal points attacked. Her first symptom was loss of vision in the right eye. A year later she had pain in the back and abdomen and constipation; an exploratory laparotomy was performed. After the operation, she immediately exhibited signs of paraplegia, and the diagnosis of poliomyelitis was advanced. She improved after six months of hospitalization and was allowed to go home. Three months later the progressive ascending paralysis returned and caused her death in six weeks.

Autopsy revealed a severe demyelinating and necrotizing process of the major portion of the spinal cord, the right optic nerve and the lower portion of the medulla. Microscopically, the important changes were demyelination of nerve fibers, destruction of axons, proliferation of neuroglia and perivascular infiltration. The process was oldest in the lumbar region of the cord and in the right optic nerve. A single microscopic lesion was discovered in the brain. The unique feature in this case was the comparatively long survival time (over two years).

Case 5.—Mrs. B. S., a 46 year old Negro woman, was admitted to the Neurological Institute on May 2, 1947, with the history of increasing motor and sensory disability of the arms and legs for two months. Two months before admission the patient experienced sudden pain in the right flank, which descended during the first week as far as the right knee but did not progress farther. One month later she noted similar pain in the left side and leg; this was accompanied with pain in the right shoulder, radiating down the arm to the elbow. During this time the patient also noticed gradually progressive numbness down the right leg. She began to have difficulty in walking, which was also progressive. Four days prior to her admission there was a sudden increase in the paresis of the right leg; the

leg became so stiff that she could not move it, and she lost all sense of control over it. At this time she also noted weakness of the right arm and hand.

Past History.—The patient had had malaria as a child. Since her marriage she had borne six living children, and four induced abortions were performed, for economic reasons, between 1926 and 1931. She was admitted to the Presbyterian Hospital in 1931 for weakness and easy fatigability, of four months' duration. Physical examination revealed poor teeth, palpable liver and a spleen enlarged 3 fingerbreadths below the left costal margin. Laboratory examination disclosed severe anemia (hemoglobin, 35 per cent; red cells, 2,080,000) and a differential smear compatible with secondary anemia. The absence of free hydrochloric acid in the gastric contents was discovered, and she was given liver extract, without a reticulocyte response. She was then given a preparation of iron, and her red cell count rose to 3,950,000, with 54 per cent hemoglobin. The diagnosis of secondary anemia following repeated pregnancies and abortions was made, and she was discharged to the outpatient clinic, after having been given contraceptive advice. A moderate anemia persisted, and she continued to complain of weakness and lassitude. Liver extract and iron were prescribed regularly for several years.

The patient was again admitted to the Presbyterian Hospital in 1937, with acute otitis media. A myringotomy was performed, and she was kept for further studies of her anemia. In spite of the most thorough laboratory and clinical studies, no cause for the anemia was found. In 1942 she was placed under thyroid medication for the second time, but she remained weak and anemic. In 1944 the patient was readmitted to the Presbyterian Hospital for weakness and anemia. After a week of intensive liver therapy, without appreciable effect, she was given two transfusions of whole blood, which brought her red cell blood count up to 3,000,000. Because the patient presented such a serious diagnostic problem and because all other means of therapy had failed, splenectomy was performed, without benefit.

The patient was seen in the eye department of the Vanderbilt Clinic in July 1946, with a complaint of blurred vision in the right eye. Vision was 20/50 in the right eye and 20/20 in the left eye. With correction of + 1.00 D. sphere, vision in the right eye was 20/20. Examination of the eyes revealed nothing pathologic. Peripheral and central fields were within normal limits. Reexamination of the eyes in February 1947 revealed no abnormality.

Examination.—The patient was thin and underweight and appeared chronically ill; she was dull, uncooperative and mentally underproductive. She showed generalized lymphadenopathy, with pronounced enlargement of the axillary nodes. The spleen had been removed; the liver was palpable 2 fingerbreaths below the costal margin and was tender. The heart evidenced mitral and systolic murmurs (probably hemic). Neurologic examination revealed spastic paralysis of the legs and weakness of the right arm. The deep reflexes were exaggerated. Sensation was impaired in all extremities. The cranial nerves were intact except for the second; the fundi showed anemic changes, with pathologic pallor of the right optic disk. Vision was not determined. The ophthalmologic consultant found pallor of both optic disks, more advanced on the right than on the left. The remainder of the physical findings were apparently normal, but cooperation was so poor that this is not certain.

Laboratory Findings.—The average blood picture, determined on many occasions, was as follows: red cells, 2,620,000; hemoglobin content, 9.3 Gm. per hundred cubic centimeters; anisocytosis, poikilocytosis, macrocytosis and occasional nucleated red cells; white cells, 5,450, with a normal differential count. Persistent reticulocytosis, with a count of 6 to 10 per cent, was found, with a rise to 53 per cent

on treatment with folic acid (pteroylglutamic acid). The urine was always normal. Serologic reactions were normal. Examination of the cerebrospinal fluid revealed clear fluid, with normal dynamics; 10 cells per cubic millimeter, all lymphocytes; 140 mg. of protein per hundred cubic centimeters, a negative Wassermann reaction, and a colloidal gold curve of 2223322100. An abundance of additional laboratory data were compiled but failed to reveal the etiologic basis of her illness.

Course.—With minor remissions, the patient became progressively worse and presented the clinical aspects of fairly rapid ascending myelitis. On one occasion she again complained of severe abdominal pain, for which no cause was found. Liver and folic acid therapy, combined with several blood transfusions, failed to ameliorate her condition or check her downhill course, or appreciably to alter the blood picture. The spinal cord level was determined to lie at the fourth cervical segment shortly before her death, on Sept. 1, 1947.

Clinical Diagnoses.—The clinical diagnoses were (1) chronic anemia, type undetermined, and (2) ascending myelitis, cause undetermined.

Autopsy.—Macroscopic Observations: External examination was noninformative. Internal examination revealed that the spleen was absent and the liver very large. Clear amber fluid and many dense adhesions were found in the pleural cavities. The lungs presented a salmon gray appearance. The parenchyma of the brain had a marked pallor; the brain stem and cerebellum appeared normal. In the thoracic segments of the spinal cord, an irregularly outlined cavity was seen in the central portion of the cord. The cord at this level was flattened and reduced in consistency. On cut section, the normal architectural markings were obscured.

Microscopic Observations: The lungs presented the typical picture of bronchopneumonia. Since the changes in the liver, stomach, bone marrow and other organs were not clearcut and are not pertinent to this paper, only the pathologic changes in the central nervous system will be presented.

Brain and brain stem. Histologically, the cerebrum and brain stem, with the exception of the lower portion of the medulla, were normal. In the lower part of the medulla, lesions similar to those to be described in the spinal cord were scattered through the parenchyma; these lesions were smaller and less severe than those in the spinal cord.

Optic nerves. The hematoxylin and eosin stain revealed a large area of degeneration in the center of the right optic nerve; the connective tissue spaces between the nerve bundles appeared wider than normal and contained numerous perivascular collections of lymphocytes. There was considerable astrocytosis in the center of this lesion. The Mahon stains showed that this lesion conformed to a large, confluent area of demyelination, the center of which was more intensely demyelinated than the periphery (fig. 8). With the Bodian stain, a considerable loss of axons could be seen in the zone of demyelination. The left optic nerve appeared normal. The optic chiasm showed diffuse demyelination on one side, the right (fig. 9).

Spinal cord. The cervical segments of the cord presented many large and small areas of necrosis and demyelination in all the white columns (fig. 10 *a*). The thoracic sections showed such extensive necrosis and dissolution of tissue that they were unidentifiable. In the upper thoracic segments (fig. 10 *b* and *c*) there was an area of cavitation in the posterior white columns, in which all the neural elements had been destroyed. There were also diffuse rarefaction of most of one lateral white column and a zone of cystic degeneration near the periphery of the cord. In the middle thoracic segments (fig. 10 *d*) the area of cavitation

surrounded the central canal and extended dorsally through the posterior white columns and ventrally into both anterior gray horns. This degeneration in the anterior gray horns was associated with total disappearance of nerve cells and glial

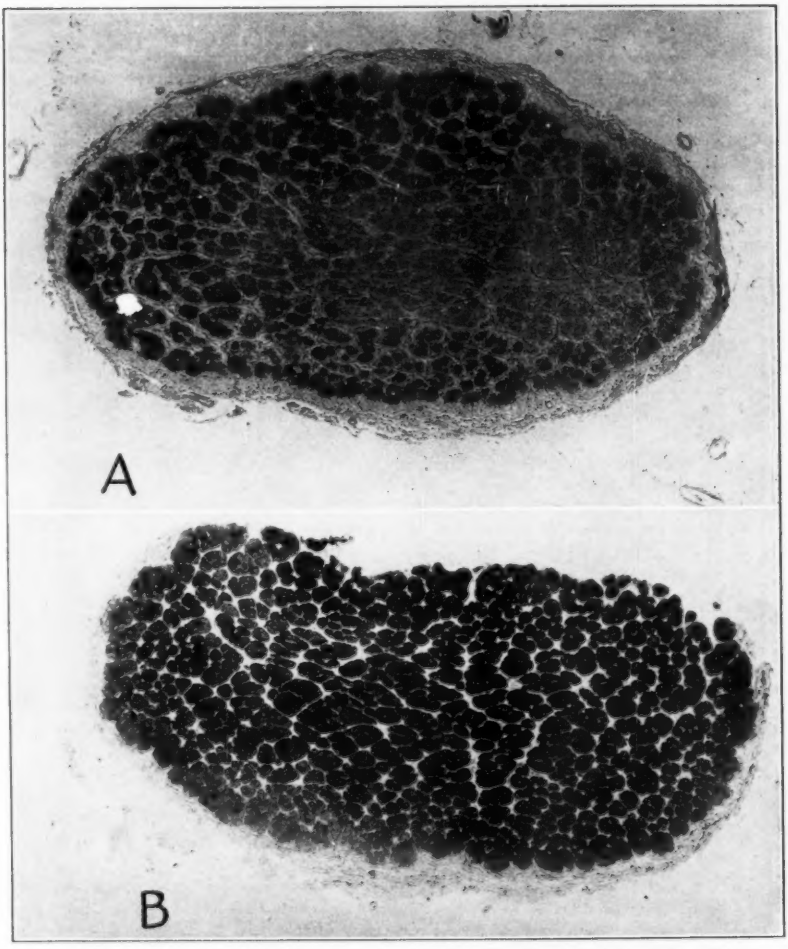

Fig. 8 (case 5).—Mahon stain of the optic nerves. (*A*) The right optic nerve shows a large diffuse focus of demyelination in the center of the nerve. (*B*) The left nerve is normal and is included for comparison.

elements, leaving a loose-meshed microcystic tissue. On one side the cystic area extended ventrally through the anterior white columns to reach the pial surface.

Posteriorly, it likewise extended in bandlike fashion across the posterior white columns to reach the pial surface near one of the dorsal roots. In the lower thoracic segments (fig. 10*e*) the cavitation was so extreme that the cord had lost its shape. Here, there was total destruction of most of the central gray matter. There were large, confluent areas of cavitation in the posterior white columns and macroscopically cystic lesions in the lateral white columns and, to a lesser degree, in the anterior white columns.

Histologically, the areas where neural elements could be recognized showed the same pathologic changes that have been described in the preceding 4 cases. The Mahon stain showed intense demyelination; the Bodian stain, a great loss of axons, and the phosphotungstic acid hematoxylin stain, considerable astrocytosis about the margins of the lesions. Perivascular lymphocytic infiltration and microglial proliferation with formation of phagocytes were seen throughout all the sections. In the most severely degenerated areas there was complete destruction of all the normal structures, leaving small cystic cavities which were bordered by partially degenerated tissue. In these zones the gray and white matter was rather loose and was composed of strands of glial tissue and persistent blood

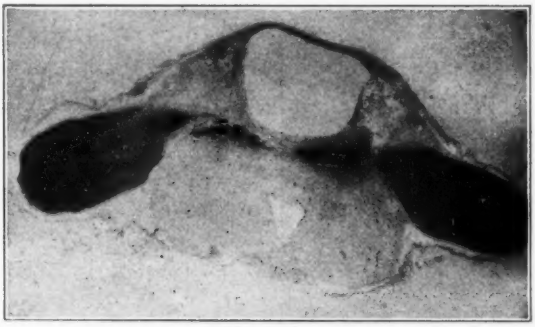

Fig. 9 (case 5).—Mahon stain of the optic chiasm, showing diffuse demyelination on one side (the right).

vessels. In the interstices of this spongy tissue were great numbers of coarsely vacuolated, lipid-laden phagocytes. There was no gliosis in any of the areas of necrosis.

The lumbar and sacral segments of the cord (fig. 10*f*) were relatively normal.

Anatomic Diagnosis.—The anatomic diagnosis was neuromyelitis optica.

Summary.—This case of long-standing anemia presented a difficult diagnostic problem. The following observations appeared significant: hepatosplenomegaly, hyperglobulinemia, anemia, leukopenia and gastric achlorhydria. Despite refractoriness to liver therapy for fifteen years, the patient did show a reticulocyte response to crude liver extract during her final hospitalization. An apparently unrelated neurologic syndrome appeared during the last four months of the patient's life; the signs and symptoms were those of an ascending myelitis, the nature of which was not determined clinically.

Necropsy revealed metaplasia of the gastric mucosa and hyperplasia of the bone marrow, such as is frequently seen in pernicious anemia. Although the early history does not fit into this classification, the terminal picture appears to do so. The patient, therefore, had two unrelated diseases.

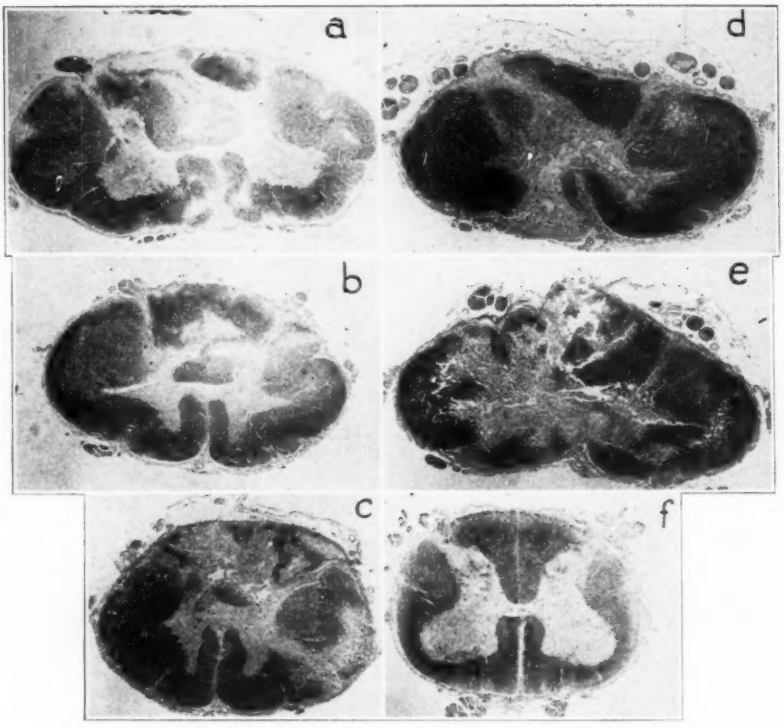

Fig. 10 (case 5).—Mahon stain of sections of the spinal cord, showing necrosis and gross cavitation of the thoracic segments: (*A*) cervical segment, (*B* and *C*) upper thoracic segments, (*D*) middle thoracic segment, (*E*) a lower thoracic segment and (*F*) a normal lumbar section.

In the central nervous system, autopsy disclosed necrotic lesions in the spinal cord and the right optic nerve; the spinal lesions were not confined to any tracts but were irregularly scattered throughout the substance of the cord. The presence of comparable changes in the spinal cord and the right optic nerve is consonant with the diagnosis of neuromyelitis optica.

CLINICAL FEATURES

Occurrence.—Approximately 200 cases of neuromyelitis optica have been reported in the literature; the number is difficult to determine because of the numerous terms employed to designate this syndrome, and especially because of the doubts existing concerning its specificity. The disease occurs in both sexes; Michaux [30] found it commoner in females and Silbermann [29] in males. The syndrome probably occurs at any age; it has been reported in children of 9 years (Berliner,[16] Walsh [17]) and in a man of 60 years (Goulden [10]). Almost all the reported cases have occurred in white persons, but this may be due to the better medical care generally available to persons of the white race. It has recently been reported in Negroes (Dolgopol,[22] Silbermann [29]) and in a Chinese (Chang [54]); 2 of the cases in this report were of Negro women. There are no known occupational factors. Heredity apparently plays no part; there are no reports of its incidence in two generations of the same family.

Onset.—Prodromal symptoms are not unusual in cases of neuromyelitis optica. Mild antecedent illnesses, usually described as sore throat, "cold," "flu," headache, fever or simply malaise, are frequently noted in the published case reports. These symptoms usually precede the onset of the visual or spinal disability by a few days to two weeks.

In its most dramatic form, neuromyelitis optica occurs in a previously healthy young adult and is ushered in by sudden, complete loss of vision in one eye, followed by loss in the other eye a day or two later. An acute onset, whether the initial symptom is visual or spinal, is nearly always seen. With regard to the temporal priority of involvement of one system over the other, there is no accord. Katz [11n] discussed this first; of his 21 patients, 15 lost vision first, 1 showed spinal involvement first and 5 showed involvement of the two systems simultaneously. According to Goulden,[10] over 80 per cent of patients evidence loss of vision as the initial symptom; Henneberg,[13d] too, reported visual loss first in about 75 per cent of cases. Beck,[12] however, found the myelitis first in 52 per cent of cases, the blindness first in 25 per cent and the two together in 23 per cent of cases. Michaux [30] stated that it is of no importance which system is involved first. All have agreed that the time interval between involvement of the two systems is variable, from hours to days to weeks.

Loss of Vision.—In most of the reported cases the visual loss was rapid, severe and monocular; the visual acuity typically falls to light perception or complete amaurosis in one day. Recently, however, a number of cases have been reported with vision reduced only to 20/200; presumably, the patients had lost their central vision. The second eye is usually involved within a matter of days, and the loss is, again, as severe and rapid as in the first eye. The amaurosis is commonly

bilateral, although cases of monocular involvement have been reported (de Lapersonne,[11a] Walsh [17] and cases 4 and 5 in this report). Walsh [58] described a few cases in which there was binocular loss of vision not followed by paraplegia; he declared that these cases represented an abortive form of neuromyelitis optica, explaining that the symptoms referable to the spinal cord may be minimal in some cases.

Ocular Fundi.—Considerable variation in the fundus picture early in this disease has been described, but only three types appear to occur commonly: (1) a normal fundus; (2) slight blurring of the margins of the disks with slight venous dilatation, and (3) acute papillitis, with edema of the disk, engorged veins, hemorrhages and exudates (Klar [41]). Later in the course of the process, optic nerve atrophy, of varying pattern, is found. Secondary optic nerve atrophy of the postpapilledema type is often described, or pallor of the disk may go on to primary optic nerve atrophy, with attenuation of the blood vessels. Michaux [30] divided his cases into two types according to the findings in the fundus: (1) retrobulbar neuritis, with a normal disk and a central scotoma (the patients with this type complain of pain, according to Michaux), and (2) papillitis, with varying degrees of severity. Michaux admitted that this division is not absolute; he stated that in many cases the process begins with the first type of optic disk and later shows frank papillitis. Goulden [10] stated that optic neuritis is almost always found and that retrobulbar neuritis is rare. Paton,[59] on the other hand, said that the characteristic lesion of neuromyelitis optica is a typical retrobulbar neuritis, with pain and loss of function. According to Paton, slight papilledema is sometimes seen but true papillitis is rare.

Visual Fields.—Many defects in the visual fields are possible and likely in neuromyelitis optica because of the irregular distribution of the lesions (Holden [60]) and because the chiasm is frequently involved. The commonest reported defect is a central scotoma, especially for colors (Fralik and DeJong,[21] and many others). The scotoma is usually bilateral, but monocular central defects are not rare. Hemianopsia was reported by Erb [3] and Holden [60]; the latter author stated that monocular hemianopsia is an important diagnostic aid in this syndrome.

58. Walsh, F. B.: Clinical Neuro-Ophthalmology, Baltimore, The Williams & Wilkins Company, 1947, pp. 783-790.

59. Paton, L.: Papilledema and Optic Neuritis: A Retrospect, Arch. Ophth. **15**:1-20 (Jan.) 1936.

60. Holden, W.: A Report of Four Cases of Acute Disseminated Myelitis with Retrobulbar Degeneration of Optic Nerves, Arch. Ophth. **40**:569-576, 1911; A Fifth Case of Acute Disseminated Myelitis with Retrobulbar Inflammation of the Optic Nerves, ibid. **43**:231-233, 1914.

Klar [41] found hemianopsia for colors, and he also described peculiar quadrantic defects with crescentic extensions. Halmajoz [61] reported a case with bitemporal hemianopsia. Concentric contraction of the peripheral fields has been reported in a number of cases, although the peripheral loss was not so severe and permanent as the central defect. Walsh [58] stated that the peripheral field becomes active before the central region in the patients who recover, and Chang [54] reported a case in which the central scotoma remained after the peripheral field had widened. In many cases of this disease the vision was so reduced that testing the fields was impractical and, further, many patients were too ill for testing. Thus, often the changes in field are not known.

Palsies of Extraocular Muscles.—Involvement of the extraocular muscles in neuromyelitis optica is uncommon but has been reported a few times. Michaux [30] found paresis of the right internal rectus muscle in his case. Walsh [58] reported 2 cases with muscular palsies: one with transient ptosis, and the other with a paretic squint that later became concomitant. McKee and McNaughton [28] described paralysis of conjugate movements of the eyes in their case. Complete ophthalmoplegias were reported by Bielschowsky,[62] Mahokian,[11k] Henneberg,[13d] Kerschensteiner [11x] and Perritt.[63] In case 3 of the present report there developed successively paralysis of convergence, paralysis of upward gaze and, finally, bilateral complete ophthalmoplegia.

Pupils.—The pupillary reactions in this disease are not characteristic; the dilatation of the pupil usually corresponds to the visual loss in the eye. In the acute stage of blindness, the pupils are widely dilated and react sluggishly, or not at all, to light. The pupillary reflexes return to the same degree as vision.

Nystagmus.—Nystagmus, so common in multiple sclerosis, is an exceptional symptom in neuromyelitis optica. When it occurs, it is assumed to have an amblyopic basis. Some authors have claimed that nystagmus has been reported only in cases in which the diagnosis was questionable. Putnam and Forster [27] described nystagmus in a case, but the diagnosis was later changed to multiple sclerosis. Michaux [30] employed the presence or absence of nystagmus as an important factor in differentiating neuromyelitis optica from multiple sclerosis.

Pain.—Pain in or about the eye is recorded in a minority of case reports. Bouchut and Dechaume [13j] reported pain in the orbits early in

61. Halmajoz: Le syndrome de Devic au début d'une sclérose en plaques, Rev. neurol. **60**:178-178, 1933.

62. Bielschowsky, A.: Myelitis und Sehnervenentzündung, Berlin, S. Karger, 1901.

63. Perritt, R. A.: Optic Neuromyelitis, Report of Two Cases, Arch. Ophth. **11**:492-497 (March) 1934.

the course of illness in their case. The typical pain of retrobulbar neuritis was prominent in the case reported by Fralik and DeJong.[21] Retrobulbar pain, associated with blurring of vision, was the earliest sign of the disease in case 2 of the present report.

Pain associated with the myelitis is a commoner symptom. It was prominent in the symptoms of cases 4 and 5 of this report. McAlpine [28] stated that pain is a common early symptom of the disease, usually referable to the back, shoulder girdles or legs.

Fever.—Low grade fever has been reported in a few cases of neuromyelitis optica. The increase in temperature is indicative of poor prognosis, according to Michaux,[30] who stated that most patients with fever die.

Cerebral Signs.—All the symptoms of neuromyelitis optica are not referable to the optic nerves and spinal cord. General and local cerebral signs are not unusual. Devic [7] himself first called attention to the cerebral symptoms, and their occurrence has recently been emphasized by Wilson.[64] Among the cerebral symptoms that have been described are jacksonian epilepsy (Jumentié and Valière-Vialeix [13g]); mental confusion (Genet and Devic [13i]); headache, vomiting and dysarthria (Cestán, Riser and Planques [30]), and aphasia (Marinesco and associates [32]). Clouding of consciousness, delirium, fainting attacks, ataxia and tremors have also been described in this disease. Paroxysmal facial neuralgia characterized 1 of the cases reported by Collins.[65] In case 3 of the present report the patient exhibited euphoria, confusion, somnolence, disorientation as to time and place, poor coordination, dysarthria and coma. Because of the frequency of cerebral signs, Barrera [66] suggested "ophthalmoencephalo-myelopathy" as a more appropriate name for the disease.

Paraplegia.—The paraplegia of neuromyelitis optica is not distinctive; the most frequently reported type is a steadily progressive ascending myelitis, with both motor and sensory loss. Landry's paralysis has been suggested as the correct diagnosis, but the sensory loss rules against this syndrome. The neurologic signs may be indicative of any of the various forms of myelitis: (1) diffuse myelitis, involving several segments of the cord; (2) complete transverse lesion, at any single level; (3) Brown-Séquard syndrome, or (4) one or more small discrete lesions, involving one or several tracts. Considering

64. Wilson, S. A. K.: Neurology, Baltimore, Williams & Wilkins Company, 1940, vol. 1, pp. 207-209.

65. Collins, J.: Acute Myelitis and Thrombotic Softening of the Spinal Cord, Rev. Neurol. & Psychiat. **10**:157-173, 1912.

66. Barrera, S. E.: Ophthalmo-Encephalo-Myelopathy: Clinico-Pathological Study of a Case, Psychiat. Quart. **6**:421-437, 1932.

the anatomic possibilities, one can readily comprehend the variability of the symptoms of the spinal cord, when both objective and subjective signs depend on the anatomic site of the lesion. Clinically, many combinations of upper and lower motor neuron lesions are seen; the deep reflexes may be lost, retained, increased or depressed. The paraplegia may be spastic or flaccid; it may change from one type to the other, or it may be spastic on one side of the body and flaccid on the other. Associated with the paralyses are all the known forms of sensory disturbance; particularly common are paresthesias, sensory loss in one leg, patchy sensory defects over the torso and extremities and a transverse sensory level in the thoracic region with complete loss below and a band of hyperesthesia above. Urinary and fecal retention or incontinence, giving rise to serious nursing problems, are seen in almost every case. The ascending paralysis eventually reaches the vital bulbar centers, and the common cause of death is respiratory paralysis. In the past, urinary infection and decubitus ulcer were prominent causes of death.

Cerebrospinal Fluid.—There has been no uniformity of opinion in regard to the spinal fluid findings. Most writers agree that the fluid is clear and that the pressure is not increased. Michaux,[30] however, stated that a slight increase in pressure was characteristic; he also expressed the opinion that increased albumin, moderate lymphocytosis and a noninformative colloidal gold curve were characteristic of Devic's syndrome. Mild pleocytosis has been reported in the majority of cases in which the spinal fluid has been examined; Popow's case [43] was unusual in that 3,000 cells per cubic millimeter were found. An increase in polymorphonuclear leukocytes in the spinal fluid was recorded by Fralik and DeJong,[21] Balser [18] and McAlpine.[25] An increase in protein, namely, the globulin fraction, has been reported by nearly every one who has examined the fluid. No abnormality has been found in the sugar of the spinal fluid, nor is the Wassermann reaction considered relevant. Colloidal gold curves are usually described as noncontributory.

Course.—Neuromyelitis optica may take one of three courses: (1) regression, to complete or nearly complete recovery; (2) one or more remissions, with an eventual fatal termination, or (3) a progressive downhill course to death. In those cases in which recovery occurs, visual acuity is usually regained before the functions of the spinal cord (McKee and McNaughton [23]). Vision may begin to improve within a week or two after the onset of the disease, and within a few weeks it may be normal. Walsh [58] stated that there is recovery of vision in the majority of cases and that he has seen only 1 case of permanent blindness. The paraplegia regresses more slowly, requiring several months before locomotion and sphincter control are regained

(Erb,[8] Seguin [4]). In many cases of recovery there are residual neurologic symptoms.

Beck [12] first drew attention to the occurrence of remissions in the course of neuromyelitis optica; he reported three complete remissions in his case and called them the first to be reported. In many cases improvement has been transient for a few days, but this is not considered a remission. Perritt [63] found that almost 50 per cent of the recorded cases have been characterized by remissions, a surprisingly high percentage in comparison with the estimates of others. In many of the cases with remissions the disease has eventually proved to be multiple sclerosis. In none of the more authentic cases with remissions has the patient lived.

In the third, and largest, group of cases, with the course progressing to death in the initial attack, a number of patients have died in a remarkably short time: Chisholm's [5] patient died in twelve days; Goulden's [10] in sixteen days; Dollfuss',[31f] in six days, and Uréchia and Veluda's,[37] in five days. Death early in the disease is usually attributed to encroachment of the ascending myelitis on the vital centers in the medulla, with ensuing respiratory paralysis. McAlpine,[25] however, stated that death more frequently results from paralysis of the phrenic nerves by a cervical lesion. Death also occurs later in the initial attack, sometimes after several months; in addition to respiratory failure, it may then be due to secondary complications, such as decubitus ulcer, ascending urinary infection or pulmonary infection. A few patients live for a considerable length of time; Dolgopol's [22] patient lived for three years and eight months.

Prognosis.—It has generally been accepted that the mortality rate of neuromyelitis optica is high, and most authors quote 50 per cent as the expected death rate. Devic [7] reported 5 deaths in his series of 17 cases. In each of Goulden's [10] series of 52 cases, 25 deaths, and in Beck's [12] series of 70 cases, 25 deaths were reported. Putnam and Forster [27] stated that the urinary infection, decubitus and respiratory failure may be prevented by mechanical means, that the present mortality rate is extremely low and that the outlook for improvement is fairly good. On the other hand, Hassin [20] stated that the pathologic process of this disease is so severe that recovery cannot be expected. It is difficult to form an accurate estimate of the mortality rate in this syndrome because the diagnosis is obviously uncertain in the cases of recovery.

Treatment.—When the etiology and pathogenesis of a disease are unknown, therapy is usually unsatisfactory, and this is certainly true of neuromyelitis optica. In the early cases all manner of medicines were used, none of which proved efficacious. Arsenic, commonly employed abroad for multiple sclerosis, is contraindicated in cases of

neuromyelitis optica, according to Brain.[67] A cure in 1 instance (Haynes[68]) was attributed to vitamin B therapy. Putnam and Forster,[27] who stated the belief that most patients can be saved, recommended good nursing care, tidal drainage for the cystitis and use of the respirator for the respiratory paralysis. In view of the new allergic theory of the causation, Silbermann[29] suggested that desensitization with histamine or allied substances may prove helpful. Surgical intervention, i. e., exploratory craniotomy, ventriculography, and even lumbar puncture, is not contraindicated in this condition, according to Walsh,[58] Woods[69] and Schaltenbrand,[70] all of whom have reported death soon after such measures were attempted. The only measures that can be recommended at present are good nursing care and proper hygienic attention to the skin, urinary and intestinal tracts and musculature; tidal drainage will eliminate, or at least reduce, deaths from urinary sepsis. There is no known specific therapy.

Summary of Clinical Features.—It is obvious from the preceding paragraphs that the literature abounds in contradictions concerning the clinical findings in neuromyelitis optica. For example, Goulden found loss of vision the initial symptom in over 80 per cent of cases, while Beck, surveying much the same group of cases, found visual loss first in only 25 per cent of cases. Likewise, Goulden stated that optic neuritis is commonly found and that retrobulbar neuritis is rare, while Paton claimed that retrobulbar neuritis was the characteristic lesion and that papillitis was rare. Therefore, in an endeavor to determine more accurately the clinical features of neuromyelitis optica, 15 of the most complete case reports published in the last twenty years were studied, mainly from an ophthalmologic point of view, and the data combined with those of the 5 cases presented in this report. Only cases in which the diagnosis was substantiated by autopsy have been selected; thus, whether or not neuromyelitis optica is a specific entity, these cases are proved examples of what is customarily considered neuromyelitis optica. The data are presented in table 1. When there was any question of interpretation (as in the visual acuity in a number of cases) the findings are given in the words of the author and are in parentheses.

The following conclusions may be drawn from these 20 case reports:

Age: Neuromyelitis optica may occur at any age but is commoner in persons between 30 and 50 years of age.

67. Brain, W. R.: Diseases of the Nervous System, London, Oxford University Press, 1947, pp. 486-516.

68. Haynes, G. W.: Neuromyelitis Optica: Report of a Case, Arch. Neurol. & Psychiat. **43**:1267-1269 (June) 1940.

69. Woods, A. G., in discussion on McKee and McNaughton,[23] pp. 136-137.

70. Schaltenbrand, G.: Hirngeschwulstähnliche Erkrankungen die reine Geschwülste sind, Ztschr. f. d. ges. Neurol. u. Psychiat. **161**:162-166, 1938.

Case No.	Author	Age	Sex	Initial Symptom	Visual Loss	Field Defect	Fundus Findings	Remissions
1	Beck [12].....	15	F	Visual	"Nearly blind, both eyes"	Bitemporal hemianopsia early; later, large central scotomas, both eyes	Secondary optic atrophy early; primary optic atrophy later	3
2	Walsh.....	9	F	Visual	Light perception, both eyes	No report	Pallor of disks and blurring of disk margins of both eyes	0
3	Fralik and DeJong [21]	31	M	Visual	Hand movements, both eyes	Large, central scotoma, both eyes	Marked optic neuritis early (both eyes); secondary optic atrophy (both eyes) later	1
4	Balser [18] (case 1)	42	F	Spinal	"Completely blind"	No report	Blurring of disk margins and hyperemia	0
5	Balser [18] (case 2)	50	F	Spinal	20/60, both eyes	Full to confrontation test, early	Blurring of disk margins	0
6	Balser [18] (case 3)	15	F	Spinal	Blind, left eye; later: 20/60 right eye; 20/80 left eye	Constriction of peripheral fields (both eyes), with a pericentral scotoma (left eye)	Pallor of disk of left eye	2
7	Dolgopol [22]	35	F Negro	Spinal	"Poor, with progressive impairment"	Normal (early)	Pallor of disks, greater in left eye	1
8	Cone, Russel and Harwood [15]	36	M Negro	Visual	"Complete blindness"	Large central scotoma (both eyes); normal peripheral fields	Pallor of disks, more in right eye; later marked optic atrophy (both eyes)	1
9	Noran and Polan	48	F	Visual	"Total blindness, both eyes"	No report	"Optic nerve atrophy"	2
10	Kohut and Richter [28]	43	F	Spinal	Right eye: no light perception; left eye: approximately normal	No field in right eye; field of left eye full to confrontation test	Right eye: acute optic neuritis, with edema, hemorrhages and exudate; left eye: slight blurring of disk margins	None
11	Hassin [20]....	33	F	Spinal	Right eye: hand movements; left eye: no light perception	No report	Right eye: mild pallor of the disk; left eye: slight blurring of the disk	1
12	Hurst, de Crespigny and Fry	46	F	Visual	Right eye: no light perception; left eye: no light perception	No report	White disks, showing optic atrophy and small vessels (both eyes)	4
13	McAlpine [14] (case 1)	24	F	Spinal	Both eyes: light perception, early; later, "blind, both eyes"	No report	"Optic atrophy, both eyes"	1
14	McAlpine [14] (case 2)	26	F	Spinal	Right eye: light perception; left eye: counting fingers	Large, central scotoma (left eye)	Blurred disk with full veins, right eye, and temporal pallor of disk, left eye: later, optic atrophy (both eyes)	2
15	Lowenberg, DeJong, and Foster	45	M Physician	Visual	No report	No report	Moderate optic atrophy (both eyes)	1
16	Stansbury, (case 1)	31	F Negro	Visual	Right eye: 20/20; left eye, 20/200	Large, central scotoma (left eye)	Right eye: normal; left eye: moderate temporal pallor	None
17	Stansbury, (case 2)	57	M	Visual	Right eye: 20/200; left eye, 20/200	Large, central scotoma (both eyes)	Blurring of disks (both eyes)	None
18	Stansbury, (case 3)	18	F	Spinal	"Poor vision, both eyes"	Patient too ill for field testing	Pallor of disks (both eyes)	None
19	Stansbury, (case 4)	29	F	Visual	"Blind, right eye"	No examination	Primary optic atrophy (right eye); normal (left eye)	2
20	Stansbury, (case 5)	46	F Negro	Spinal	Right eye: 20/50; left eye; 20/20 (early)	Fields normal (early)	Pallor of optic disk (right eye); normal (left eye)	None

Length of Disease	Spinal Fluid	Course of Paraplegia
13 mo.	4 cells/cu. mm.; protein 0.04 mg./100 cc.; weak syphilitic curve in Lange test	Back pain; weakness and numbness of legs; paralysis of legs; incontinence of urine and feces; improvement; recurrence of previous symptoms; paresthesias; complete paralysis of arms and legs; sensory loss to sixth thoracic level; hyperactive reflexes; respiratory failure; death
6 wk.	Normal pressure and dynamics; 27 cells/cu. mm. (80% polymorphonuclear cells, 20% lymphocytes); sterile culture; nonprotein nitrogen 20 mg., chlorides 712 mg. and sugar, 58 mg./150 cc.	Tingling and weakness of feet; flaccid paralysis of legs; loss of sphincter control; patchy sensory loss; variable reflexes; weakness of arms; loss of sensation below nipple line; dermatographia; hypesthesia of face; apathy and drowsiness; flaccid paralysis of arms; diaphragmatic respiration; dysarthria; dysphagia; death
2 mo.	Pressure 230 mm. water; 10 lymphocytes/cu. mm.; globulin 4+; colloidal gold curve 0001110000	Numbness of feet; staggering; weakness and numbness of legs; hyperactive reflexes; constipation; inability to walk; urinary incontinence and fecal retention; sensory loss below seventh thoracic level; urinary infection; decubitus ulcers; respiratory failure; death
9 wk.	184 cells/cu. mm. (15% polymorphonuclear cells); protein content, Wassermann reaction and gold curve normal	Asthenia and pains in legs; pain in shoulders and neck; rigidity of neck; incontinence of urine and feces; sensory loss to sixth thoracic level; abnormal reflexes; respiratory difficulty; death
7 wk.	58 cells/cu. mm.; protein 110 mg./100 cc.; Wassermann reaction negative; colloidal gold curve normal	Numbness of feet; weakness of legs; numbness and weakness of left arm; incontinence of urine and feces; poor coordination; progressive sensory loss; abnormal reflexes; death from respiratory paralysis
20 mo.	15 cells/cu. mm.; protein 40 mg./100 cc.; Wassermann reaction negative; colloidal gold curve normal	Numbness and paresis of right leg and then of the left leg; weakness of left hand; loss of deep reflexes; loss of sensation below umbilicus; gradual deterioration to death
44 mo.	20 cells/cu. mm.; slight increase in globulin; sugar normal; Wassermann reaction negative; colloidal gold curve 0112332100	Numbness of legs; unsteady gait; numbness and paralysis from waist down; incontinence of urine and feces; abnormal reflexes; sensory loss to tenth thoracic level; improvement; return of previous sight; spastic paraplegia; gradual deterioration; death from pneumonia
8 mo.	Pressure 170 mm. water; 7 cells/cu. mm.; globulin reaction negative; protein 113 mg./100 cc.; reaction for lead positive	Tingling, weakness and numbness of legs; urinary retention and constipation; patchy hypesthesias and hyperesthesias; inability to stand; loss of reflexes in legs; paralysis of legs; ascending sensory loss; death
20 mo.	Pressure 7-14 mm. Hg; 4 cells/cu. mm.; protein 45 mg./100 cc.; sugar 60 mg.; colloidal gold curve normal	Numbness of feet; sensory loss to fifth thoracic level; paralysis of legs; improvement; numbness of legs; complete motor and sensory paraplegia; bladder and rectal incontinence; areflexia in lower extremities; progressive motor and sensory loss; diaphragmatic breathing; death
6 wk.	Pressure 140 mm. water, normal dynamics; 185 cells/cu. mm.; 70% polymorphonuclear cells, 30% lymphocytes; protein 241 mg./100 cc.; Wassermann reaction negative; culture sterile	Numbness of thighs; burning sensations of feet and ankles; ascending numbness; weakness of knees; inability to stand; flaccid paralysis of legs with areflexia; sensory loss to waist; stiff, painful neck; sensory loss to nipple line; weakness of arms; loss of sensation in arms; diaphragmatic respiration; death
14 mo.	No report	Pains, weakness and numbness in right leg and then in the left leg; ascending numbness to breasts; paralysis of legs; improvement; paralysis of legs again; anesthesia to umbilicus; automatic bladder; paralysis of right arm, then of left arm; constipation; restlessness and disturbed sensorium; death
20 mo.	20 lymphocytes/cu. mm.; protein 70 mg./100 cc.; increased globulin; colloidal gold curve 1112221000; Wassermann reaction negative	Girdle pains; urinary retention; lumbar pain, spreading to buttocks, thighs and legs; loss of strength and sensation in legs; hyperreflexia, then areflexia; urinary incontinence; constant pain; sensory loss to nipple line; delirium; urinary sepsis, death
18 mo.	4 lymphocytes/cu. mm.; protein 0.08 mg./100 cc.; Wassermann reaction negative; colloidal gold curve 1111222100	Pain in shoulders; paresthesias and weakness of legs; urinary retention; paralysis of abdominal and intercostal muscles; paralysis of legs; variable reflexes; loss of sensation to fourth thoracic level; fecal incontinence; improvement; abdominal pain; paralysis of legs again; loss of sphincter control; paresthesias in arms and legs; paresis of hands and wrists; decubitus ulcers and cystitis; death
26 mo.	Pressure 130 mm. water; 2 cells/cu. mm.; protein 0.025 mg./100 cc.; colloidal gold curve normal	Pain in shoulders; numbness and weakness of legs and abdomen; urinary retention; sensory loss to sixth thoracic level; drowsiness; pain and paresis of arms; complete flaccid paralysis of legs and arms; diaphragmatic breathing; improvement; urinary infection and incontinence; improvement; pain in neck; incontinence; paresis and sensory loss again; vomiting; respiratory paralysis; death
14½ mo.	30 lymphocytes/cu. mm.; otherwise normal	Paresthesias of right arm, leg and abdomen; Horner's syndrome on right; spastic paralysis of right arm and leg; hyperreflexia; patchy anesthesia; hiccups; dysuria; improvement; parethesias; paralysis of left arm and leg; respiratory difficulty; overflow incontinence; painful paresthesias; death from respiratory paralysis
3 mo.	Normal pressure and dynamics; 10 cells/cu. mm.; globulin 1+; protein 56 mg., sugar 62 mg. and chlorides 702 mg./100 cc.; Wassermann reaction negative; colloidal gold curve 1233332100	Numbness and weakness of right arm and leg; numbness of left leg and foot, then of left hand; hyporeflexia of arms and hyperreflexia of legs; hypesthesia of all extremities; urinary retention; excessive perspiration about head and neck; marked paralysis of all four limbs; respiratory paralysis; death
5 mo.	Normal pressure and dynamics; no cells; protein 60 mg., sugar 94 mg. and chlorides 681 mg./100 cc.; gold curve 1111100000; Wassermann reaction negative	Staggering; poor coordination; hyperreflexia; weakness of arms and legs; falling; tingling sensation in arms and legs; noise in ears; dizziness; ataxia; increasing weakness of extremities; reduced pharyngeal reflex; deviation of uvula; respiratory embarrassment; death
6 wk.	Normal pressure and dynamics; 9 cells/cu. mm.; protein 68 mg. and sugar 65 mg./100 cc.; gold curve 1100000000; Wassermann reaction negative; cultures sterile	Weakness and numbness of left arm and leg; unsteady gait; slurring of speech; euphoria and confusion; somnolence; weakness of right arm and leg; disorientation; loss of sensation on right; hyperreflexia; incontinence of urine and feces; bilateral third and sixth nerve palsies; left hemiplegia with spastic arm and flaccid leg; right hemiparesis; complete bilateral ophthalmoplegia; coma; decerebrate posture; respiratory embarrassment; death
2 yr.	Normal pressure and dynamics; 86 cells/cu. mm. (99% lymphocytes); protein 240 mg., sugar 40 mg. and chlorides 701 mg./100 cc.; Wassermann reaction negative and gold curve normal	Pain in back and abdomen; constipation; weakness of legs, with areflexia; improvement; paresthesias of abdomen and legs; sensory loss to breasts; urinary retention; constipation; pain in shoulders and neck; numbness and paresthesias in arms; areflexia in legs and hyperreflexia in arms; respiratory difficulty; death
6 mo.	Normal pressure and dynamics; 10 lymphocytes/cu. mm.; protein 140 mg./100 cc.; Wassermann reaction negative; cultures sterile; gold curve 2223322100	Pain in right flank; numbness and weakness of right leg, then of right arm and hand; pain in left leg and elbow; numbness of left leg; difficulty in walking; progressive paralysis of all four limbs; severe abdominal pain; vomiting and prostration; respiratory paralysis; death

Sex: The disease appears to be commoner in persons of the female sex; of these 20 patients, 16 were female.

Race: The disease is not limited to persons of the white race; 4 of the 20 patients were Negroes.

Initial Symptom: The incidence of the initial disability was distributed equally between the visual system and the spinal cord in this series of cases; in 50 per cent loss of vision, and in 50 per cent neurologic symptoms were first.

Loss of Vision: Severe binocular loss of vision is characteristic of this syndrome. The degree of visual loss varied in the 20 cases as follows:

Binocular loss	No. of Cases
Blind	5
"Nearly blind"	1
Light perception	3
Hand movements	1
20/200	1
20/60 to 20/200	3
Monocular loss	
"Blind"	3
20/200	1
20/50	1
No record of the visual acuity	1

Ophthalmoscopic Appearance: Terminally, in the majority of cases of neuromyelitis optica, optic nerve atrophy is exhibited. Of the 16 cases of this series in which the diagnosis of optic nerve atrophy was made, primary or secondary optic atrophy was described in 5; of the remaining 11 cases, the diagnosis was simply "optic nerve atrophy" in 6 and "pallor of the disks" in 5. Of the 16 cases with optic nerve atrophy, the lesions were binocular in 12. The diagnosis in the remaining 4 cases would appear to be optic neuritis, which was binocular in 3 cases.

Unfortunately, the descriptions of the fundi were too inaccurate for careful ophthalmologic analysis. Most of the patients were not examined early in their illness, and none of the case reports contain detailed, repeated observations. In addition, most of the examinations were reported by neurologists who are not accustomed to classify optic nerve atrophy according to ophthalmologic usage.

Visual Fields: Either central scotoma or loss of the entire field appears to be the characteristic field change. Large central scotomas were demonstrated in 6 cases in this series, being binocular in 4 cases and monocular in 2. Five patients were blind, and so presumably had no field, and 1 patient was blind in one eye. General constriction of the peripheral fields with a pericentral scotoma in one eye was recorded in 1 case. The fields were reported to be normal in 1 case. There was no description of the field in 6 cases.

Remissions: Remissions occurred in about half of the cases in this series (12 cases, or 60 per cent). The distribution of remissions was as follows:

No. of Remissions	No. of Cases
1	6
2	4
3	1
4	1
None	8

Length of the Disease: According to the experience of these 20 patients, a person with neuromyelitis optica is likely to die in the first three months of his illness or at some time in the second year. Seven patients in this group died within three months, and 8 died in the second year of the process.

Paraplegia: The characteristic neurologic feature in this series of cases was the variability of the clinical picture. There appears to be no pathognomonic neurologic sign or symptom in this syndrome. The initial neurologic complaint was referable to the legs in 11 cases, and the initial symptom was sensory in 13 cases. Pain was a prominent feature in 10 of the cases. The deep reflexes were always variable. The paralyses were sometimes flaccid and sometimes spastic. Cerebral symptoms were noted in 7 cases. Incontinence of the bladder and rectum was specified in 15 case reports. Respiratory paralysis was the cause of death in 14 cases. Death was due to pneumonia in 1 case, and the cause of death was not clear in 5 cases. The only consistency in the neurologic symptomatology was the ascending nature of the process; in all 20 cases, including those in which the initial symptom was located elsewhere, the legs were involved early and the rest of the body in an irregular, but generally ascending, pattern.

Spinal Fluid: There is no change in the spinal fluid characteristic of neuromyelitis optica.

If these 20 cases are representative of neuromyelitis optica, one may conclude that this disease occurs more commonly in females in the first half of life and is ushered in by either optic or spinal disability. Ophthalmologically, binocular loss of vision, optic nerve atrophy and field changes are characteristic. Neurologically, variability is the rule in this rapidly ascending myelitis. No laboratory findings are peculiar to the disease. Remissions are seen in about half the cases. In the fatal cases death usually occurs within two years.

(To Be Concluded)

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Graduate Training in Ophthalmology.—The University of Toronto Faculty of Medicine is offering a postgraduate course in ophthalmology extending over three years. The first year, on a fellowship, the value of which is approximately $1400, the student spends in one of the basic sciences of ophthalmology, and the second and third years are spent in the intern service of one or more of the university teaching hospitals. Approximately four hours a week of didactic teaching are arranged for the students by members of the staff. On Saturday mornings staff ward rounds are made at the Toronto General Hospital and are attended by the interns from the other teaching hospitals. A sound knowledge of neurology and metabolic diseases is desirable. The fee for instruction is $50 a year, payable to the chief accountant, University of Toronto.

An application for appointment may be made to the Professor of Ophthalmology, University of Toronto Faculty of Medicine. Appointments are made in December, to commence July 1.

Course in Histopathology of the Eye, New York Eye and Ear Infirmary.—The schedule of the three months' course in histopathology of the eye at the New York Eye and Ear Infirmary, given by Dr. Brittain F. Payne and associates, has been rearranged to last one week from Jan. 9 to 14, 1950.

The course will be given in the afternoons and will consist of the same number of hours of instruction and of the same material as those previously allotted. Further information may be obtained by writing to the Post-Graduate Division of the New York University College of Medicine.

Postgraduate Course in Ophthalmology, University of California Medical School.—The University of California Medical School announces a postgraduate course in ophthalmology for qualified physicians, Sept. 12 to 17, 1949, to be given at the University of California Medical Center, San Francisco, under the sponsorship of the University Extension (Medical Extension), University of California. Complete program and further details may be obtained from Dr. Stacy R. Mettier, head of postgraduate instruction, Medical Extension, University of California Medical Center, San Francisco 22.

Transactions of the American Ophthalmological Society.—The *Transactions,* published in book form, may be purchased by advance subscription. The current volume contains theses of applicants for membership and the scientific papers given at the eighty-fifth annual meeting in June 1949. The price is $12. Copies may be procured by order to the editor, Dr. Wilfred E. Fry, 1930 Chestnut Street, Philadelphia. Orders must be in by December 1.

SOCIETY NEWS

Joint Meeting of North Carolina Eye, Ear, Nose and Throat Society and South Carolina Society of Ophthalmology and Otolaryngology.—A joint meeting of the two societies will be held at the Hotel Poinsett, Greenville, N. C., Sept. 12 to 15, 1949. The program follows:

Dr. A. D. Ruedemann, professor of ophthalmology, Wayne University, Detroit:
- Differential Diagnosis of Unilateral and Bilateral Ocular Protrusion
- End Results with New Radium D Applicator as Compared with Results with Radon
- Ocular Manifestations of Allergy

Dr. Peter C. Kronfeld, department of ophthalmology, Illinois Eye and Ear Infirmary, University of Illinois, Chicago:
- Differential Diagnosis of Acute Glaucomas
- Prognosis of Retinal Detachment
- Newer Drugs in Ophthalmology

Dr. Rudolph Aebli, professor of ophthalmology, New York University, New York:
- Relation of Muscle Imbalances to Palpebral Aperture and Pseudoptosis
- Principles of Ocular Muscle Surgery
- Congenital Muscle Anomalies

Dr. Carl C. Johnson, associate, department of ophthalmology, Harvard Medical School, Boston:
- Surgical Treatment of Ptosis
- Diagnosis and Surgical Treatment of Glaucoma

Dr. Preston C. Iverson, New York:
- Neoplasms of Lip, Nose and Eyelid and Plastic Repair of the Lesions
- Congenital Deformities of the Face

Dr. Louis H. Clerf, Philadelphia:
- Cough Viewed from the Otolaryngologic Standpoint
- Surgical Treatment of Paralysis of Larynx
- Malignant Neoplasms of Larynx

Dr. Kenneth M. Day, Pittsburgh:
- Clinical Management of Deafness
- Ménière's Disease

Dr. Russell A. Sage, Indianapolis:
- Diseases of the Mouth and Tongue

Ophthalmological Society of Egypt to Award Prize.—The Ophthalmological Society of Egypt, to encourage scientific work in ophthalmology, has decided to grant a prize, to the value of 20 Egyptian liras, for the most valuable contribution presented before the Annual Congress of the Society. The competition is open to all members of the Society of less than twenty years' practice. The subject is left open. Articles must be received in Cairo not later than the beginning of December. Further details may be secured from the honorary secretary, Ophthalmological Society of Egypt, Dar El Hekma, 42 Kasr El Ainy Street, Cairo, Egypt.

Officers of New York Society of Clinical Ophthalmology for 1949-1950.—The officers elected for the coming year are: president, Dr. Sidney A. Fox; vice president, Dr. Samuel Gartner; recording secretary, Dr. Bernard Kronenberg; corresponding secretary, Dr. Leon H. Ehrlich; treasurer, Dr. Edward Saskin; librarian, Dr. Howard Agatson.

The retiring president, Dr. Benjamin Esterman, was elected to the advisory council.

UNIVERSITY NEWS

Appointments by the University of Toronto Faculty of Medicine.—The University of Toronto Faculty of Medicine has appointed Dr. H. L. Ormsby as clinical teacher in charge of bacteriology of the eye, and Dr. O B. Richardson, clinical teacher in charge of pathology of the eye, in the department of ophthalmology.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Cornea and Sclera

CORNEAL TRANSPLANTATION, R. T. PATON, J. M. MCLEAN, R. CASTROVIEJO, A. E. MAUMENEE and W. KORNBLUETH, Am. J. Ophth. **31:** 1365 (Nov.) 1948.

This is a symposium in which Paton discusses selection of cases; McLean, technics; Castroviejo, complications, and Maumenee and Kornblueth, physiopathology. The last is in the form of a statistical study.

W. S. REESE.

TREATMENT OF ROSACEA KERATITIS. J. JIRMAN, Ann. d'ocul. **181:** 475 (Aug.) 1948.

The author reviews the various ocular manifestations of keratitis rosacea and the etiologic factors suggested for this important disease of the eyes. A case of keratitis rosacea occurring in both eyes of a woman aged 54 is described in detail. She received a therapeutic trial of practically every means ever suggested for keratitis rosacea, i. e., autohemotherapy and use of riboflavin antihistamic drugs and hormones, without control of the corneal disease. Finally, one eye was covered with a conjunctival flap. There were immediate regression of symptoms and gradual objective improvement. The other eye showed no improvement until it was covered with a similar flap.

If the flap retracts within three to four days, little improvement is noted. The sutures holding the flap should be deep, so that the flap covers the cornea for ten to fifteen days. This permits the conjunctiva to take an active part in the healing of the cornea.

P. R. MCDONALD.

Glaucoma

PRESSOR TEST FOR GLAUCOMA. G. T. STINE, Am. J. Ophth. **31:** 1203 (Oct.) 1948.

Stine found the pressor test safe and easily performed for the detection of wide angle glaucoma but completely unreliable in the detection of narrow angle glaucoma.

W. S. REESE.

ELLIOT TREPHINING OPERATION FOR GLAUCOMA. R. E. MEEK, Am. J. Ophth. **31:** 1232 (Oct.) 1948.

Meek recommends use of Tenon's capsule as part of the bleb, and he modifies the operation by making an incision at the limbus so that corneal splitting is facilitated.

W. S. REESE.

ALLERGY IN GLAUCOMA. C. BERENS, L. J. GIRARD and E. CUMMINGS, Ann. Allergy **5:** 526 (Nov.-Dec.) 1947.

Three cases of chronic simple glaucoma are presented in which the usual treatment with miotics and attempted desensitization with autogenous bacterial antigens had been supplemented by the removal from the diets of food allergens, as determined by the pulse-diet method. In 1 case, the hitherto uncontrolled tension was brought under control apparently only after the institution of an allergen-free diet. In the other 2 cases, surgical and medical treatment controlled the glaucomatous hypertension but failed to check progressive loss of visual fields. The fields showed striking improvement after the institution of an allergen-free diet. The authors conclude that, while the findings are suggestive, it is inadvisable to draw definite conclusions.

W. ZENTMAYER.

Experimental Pathology

OBSERVATIONS ON EXPERIMENTAL PNEUMOCOCCAL INFECTION OF THE RABBIT'S CORNEA AND ON THEIR TREATMENT WITH PENICILLIN. J. MACASKILL and M. WEATHERALL, Brit. J. Ophth. **32:** 892 (Dec.) 1948.

The article reports further observations on the lesions produced by intracorneal inoculation of the same and other strains of pneumococci and on the effect of penicillin administered subconjunctivally on these lesions in animal experimentation. The following summary is supplied: The responses of rabbits to intracorneal injections of various strains of pneumococci are described. The lesions were variable, but with one or two strains keratitis, iritis and hypopyon were produced with sufficient constancy to permit the evaluation of therapeutic procedures. Penicillin was found to be more effective when administered subconjunctivally than subcutaneously, and by the former route completely to arrest infection when 1,000 units per kilogram of body weight were given at twelve hour intervals.

W. ZENTMAYER.

General

PSYCHOSOMATIC INTERRELATIONSHIPS IN OPHTHALMOLOGY. D. O. HARRINGTON, Am. J. Ophth. **31:** 1241 (Oct.) 1948.

Harrington decries the lack of interest manifested by ophthalmologists in psychosomatic medicine and discusses five ocular conversion symptoms and an equal number of ocular vegetative neuroses.

W. S. REESE.

TREATMENT OF DELAYED POSTOPERATIVE FORMATION OF THE ANTERIOR CHAMBER. V. A. BYRNES, Am. J. Ophth. **31:** 1261 (Oct.) 1948.

Byrnes believes that no anterior chamber should be allowed to remain flat more than five days after cataract extraction. For flat chambers he recommends inspection of the wound and cauterization of the wound with a heated squint hook or use of a diathermy needle, such as that employed in treatment of retinal detachments. He reports 5 cases.

W. S. REESE.

ON NEW TYPES OF OCULAR DISEASES. A. FUCHS, Am. J. Ophth. **31:** 1273 (Oct.) 1948.

Among new types of ocular disease Fuchs includes divided nevi, Sjögren's syndrome, Behcet's syndrome, keratitis parenchymatosa linearis, neuritis syphilitica papulosa, myopia inversa, choroiditis proliferens and neuritis retrobulbaris maligna. W. S. REESE.

Injuries

FRIEDLÄNDER'S BACILLUS INFECTION FOLLOWING PERFORATING WOUND OF ORBIT. W. J. CRAWFORD, Am. J. Ophth. **31:** 1293 over some of the test letters.

Crawford reports a case of infection of the orbit with Friedländer's bacillus following a perforating injury which improved after removal of a foreign body and treatment with streptomycin. W. S. REESE.

TREATMENT OF SUPERFICIAL CORNEAL INJURIES. A. R. SHERMAN, Am. J. Ophth. **31:** 1467 (Nov.) 1948.

Sherman remarks the slowness of healing of certain superficial traumatic corneal lesions. For these he uses a binocular bandage to encourage epithelial regeneration. He briefly reports 20 cases.

W. S. REESE.

Neurology

INTRA-OCULAR PHAKOMATA—A REPORT OF THREE CASES. R. F. LOWE, Brit. J. Ophth. **32:** 847 (Nov.) 1948.

Three cases of intraocular phakoma are recorded.

The appearance of the retinal phakomas in the 2 patients with Bourneville's disease (tuberous sclerosis) was so characteristic that diagnosis could have been made if these had been the only stigmas found. The diagnosis of the probable choroidal neurofibroma depends on the presence of numerous, widespread neurofibromas readily identified elsewhere, with bilateral secondary optic nerve atrophy, epilepsy and amentia. On appearances alone it could not be differentiated from other choroidal tumors. W. ZENTMAYER.

Ocular Muscles

OPERATIVE TREATMENT OF VERTICAL TROPIAS. B. F. PAYNE, Am. J. Ophth. **31:** 1217 (Oct.) 1948.

Payne concludes that planned operations for the relief of vertical trophias are successful if the diagnosis is correct. A clear understanding of the muscle or muscles involved is necessary, and the type and extent of operation are determined by the points brought out in the complete examination. W. S. REESE.

INFLUENCE OF TORSIONAL MOVEMENTS ON THE AXIS OF ASTIGMATISM. S. VAN WIEN, Am. J. Ophth. **31:** 1251 (Oct.) 1948.

Van Wien discusses the influence of torsional movements on astigmatism. He suggests a test for cyclophoria in all cases of high astigma-

tism and, if the condition is present, the determination of the astigmatic axes binocularly by means of polaroid filters in the trial frame and over some of the test letters. W. S. REESE.

Physiology

THE RESPIRATION OF THE STORED CORNEA. T. D. DUANE, Am. J. Ophth. **31:** 1400 (Nov.) 1948.

Duane draws the following conclusions from his study of bovine corneas:

1. Respiration studies have been made on bovine corneas stored from one and one-half hours to sixty days, under five conditions: intact eyes stored (1) above Ringer's solution at 4 C. and (2) under oil at 15 C.; isolated corneas stored in 5 per cent and 35 per cent solution of formaldehyde U. S. P., (3) in the "deep freeze" at 40 C. and (4) in the "deep freeze" after quick freezing.

2. The oxygen consumption rate (QO_2) stored over Ringer's solution or under oil at low nonfreezing temperatures remains normal for seven days. The respiratory rate falls to one-fifth normal in ten to twelve days under these conditions.

3. Solution of formaldehyde U. S.P. inhibits respiration of the cornea immediately, and this is apparently irreversible.

4. The oxygen consumption of the cornea is reduced immediately to one-fifth normal by quick freezing and drops to this level within four days when kept at — 40 C. There is no further respiratory depression up to sixty days of storage. W. S. REESE.

EFFECT OF MUSCULAR EXERCISE ON DARK ADAPTATIONS. J. P. WENDLAND, Am. J. Ophth. **31:** 1429 (Nov.) 1948.

Wendland found that light exercise had essentially no effect on the rate of dark adaptation but that heavy exercise produced a tendency to elevation of the thresholds of the total dark adaptation curve and caused a decided biphasic response in final rod thresholds.

W. S. REESE.

Retina and Optic Nerve

OPTIC NEUROPATHIES: A SIMPLIFIED CLASSIFICATION AND OUTLINE FOR ETIOLOGIC DIAGNOSIS. A. C. WOODS, Am. J. Ophth. **31:** 1053 (Sept.) 1948.

Primarily from the etiologic viewpoint, Woods divides diseases of the optic nerve into two general classes: (1) degenerations of the optic nerve secondary to ocular disease, circulatory failure, local malformations and local accidents or infections and degenerations, with a hereditary basis, and (2) diseases of the optic nerve due to disease of the central nervous system, intracranial anomalies, systemic disease and various intoxications. A concise description of the various disorders of the optic nerve is given. The author insists that the ophthalmologist manage the case until its exact nature is decided on. W. S. REESE.

HISTOLOGY OF THE RETINA IN A CASE OF TAY-SACHS'S DISEASE. J. A. MACMILLAN, Am. J. Ophth. **31:** 1567 (Dec.) 1948.

MacMillan reports a case of amaurotic familial idiocy, including the pathologic changes. He suggests that one look for metabolic abnormalities and states the belief that the abnormal material in the ganglion cells is the result of an inherited metabolic dyscrasia.

W. S. REESE.

SCLERAL RESECTION IN THE TREATMENT OF RETINAL DETACHMENT: A PRELIMINARY REPORT. S. PHILPS, Brit. J. Ophth. **32:** 811 (Nov.) 1948.

Philps reports 2 cases of retinal detachment successfully treated by scleral resection. A man aged 59 had a detachment of the retina following a blow on the right eye. After two diathermy operations the detachment was total. In the left eye a cataract had developed ten years previously. An intracapsular extraction was done and the retina was found to be detached. A diathermy operation was performed but was unsuccessful. Later a scleral resection was made. Five months later the retina was still in place; "the field was quite good," and vision was 5/60. A child 10 years of age had a detachment of the retina following a blow. A diathermy operation resulted in replacement of the retina, but nine months later a total detachment occurred. A scleral resection was then done and resulted in replacement of the retina. On last examination vision was "poor," as the macula had been involved in the detachment.

W. ZENTMAYER.

Therapeutics

USE OF BACITRACIN IN OCULAR INFECTIONS. J. G. BELLOWS and C. J. FARMER, Am. J. Ophth. **31:** 1211 (Oct.) 1948.

Bellows and Farmer found bacitracin well tolerated by the intact eye when applied topically as a fine powder or in saline solutions containing 1,000 to 5,000 units per cubic centimeter. The former solution did not appreciably delay regeneration of corneal epithelium, but the latter solution definitely retarded epithelial regeneration, with subsequent vascularization and scarring of the cornea. Bacitracin penetrated the injured and inflamed cornea, but not the normal cornea. Experimentally produced infections of the cornea and vitreous with a bacitracin-sensitive hemolytic Staphylococcus aureus were prevented when treated sufficiently early with bacitracin.

W. S. REESE.

ANTISTINE IN OCULAR ALLERGY. P. HURWITZ, Am. J. Ophth. **31:** 1409 (Nov.) 1948.

Hurwitz briefly reviews the antihistaminic clinical drugs and presents 50 cases of ocular allergy and their response to antistine® (2-phenylbenzylaminomethyl-imidazoline), a new antihistaminic ophthalmic solution. He advocates its use.

W. S. REESE.

Trachoma

THE ANTITRACHOMA CAMPAIGN IN JEWISH SCHOOLS OF JERUSALEM. B. MIRENBURG, Am. J. Ophth. **31:** 1289 (Oct.) 1948.

Mirenburg describes the antitrachoma campaign in Jerusalem and gives tables showing the incidence, distribution and control of trachoma.

W. S. REESE.

INVOLVEMENT OF THE CANALICULI IN TRACHOMA. A. TZAREVA, Vestnik oftal. **26:** 34, 1947.

Inflammation of the canaliculi is considered as a latent and recurrent source of trachomatous infection and as threatening to the corneal infection.

Tzareva examined all trachomatous patients of the Kirghiz eye clinic during the period from 1938 to 1945, paying special attention to infection of the canaliculi. In 53 patients, or 5.7 per cent, dacryocanaliculitis was observed, an enlarged punctum being usually present. In all these patients, the course of the trachomatous process as a rule was severe, with extensive pannus, corneal ulcers and, consequently, decreased vision in all eyes, with blindness in some. The method of treatment consisted in incising the canaliculi; this led to disappearance of the infection in 37 patients, while extirpation of the sac had to be resorted to in others. Thus, Tzareva urges the systematic examination of the canaliculi in order to prevent a focus of reinfection in trachomatous patients.

O. SITCHEVSKA.

Tumors

MELANOBLASTOMA OF THE HUMAN CHOROID CULTURED IN VITRO: REPORT OF FOUR CASES. F. VRABEC, Ophthalmologica **115:** 129 (March) 1948.

Using the method of Carrel, the author obtained tissue cultures of 4 melanoblastomas of the human choroid and demonstrated the invasion of the tumor cells into the retinal tissue.

H. P. KIRBER.

A RARE CASE OF MULTIFORM GLIOBLASTOMA SPONGIOBLASTOMA OF THE RETINA. A. LOTIN, Vestnik oftal. **26:** 65, 1947.

A child aged 2 years was brought into the clinic, where the diagnosis of a cataract of the right eye was made. Five months later iritis developed in that eye, and on the next visit, three months later, there was marked buphthalmos with a questionable tumor of the eye. Enucleation was performed, and microscopic examination revealed a rare histologic picture. There were characteristic degenerative pseudorosettes, with radial accumulation of the nuclear cells around the necrotic centers; anomalous giant cells and protoplasmic bodies with intensively stained nuclei. Young embryonic cells were evident. This picture was one of multiform spongioblastoma, or glioblastoma, of the retina, which Cushing found to constitute 30 per cent of gliomas of the brain.

O. SITCHEVSKA.

DIFFUSE MALIGNANT MELANOMA OF THE IRIS. S. RICHARDSON, Am. J. Ophth. **31:** 1223 (Oct.) 1948.

Richardson discusses malignant melanoma of the iris and reports 2 cases. He considers immediate enucleation the treatment of choice and does not think exenteration necessary.

W. S. REESE.

OSTEOMA INVOLVING THE ORBIT. F. W. NEWELL, Am. J. Ophth. **31:** 1281 (Oct.) 1948.

Newell presents 7 cases of osteoma, the tumor arising in the ethmoid sinus in 2, in the sphenoid bone in 1 and in the frontal sinus in 4. The growths in the ethmoid sinus were removed, but the sphenoidal osteoma was considered inoperable and caused death from internal hydrocephalus.

W. S. REESE.

Society Transactions

EDITED BY DR. W. L. BENEDICT

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Perce DeLong, M.D., *Chairman*

M. Luther Kauffman, M.D., *Clerk*

Regular Meeting, April 22, 1948

Heteroplastic Grafts: Presentation of a Case. DR. O. A. CAPRIOTTI (by invitation) and DR. EDMUND B. SPAETH.

The patient presented was a 15 year old boy from the service of Dr. Spaeth at Wills Hospital on whom successful isografts had been performed for ectropion of the four eyelids complicating ichthyosis congenita.

He was an only child, and there was no history of abnormalities of the skin on either side of the family. Two weeks after his birth, a red rash appeared over the entire body, and this was soon followed by a generalized scaly condition of the skin. The diagnosis of ichthyosis congenita was established as a child, and he was presented by Dr. John Ludy at a dermatologic meeting.

Epiphora started at about the age of 8 years, and ectropion of the lids was rather severe at the age of 10. About four years previously the patient had been treated for one and a half years for a corneal ulcer of the left eye. Prior to admission he had been treated for three months as an outpatient for a corneal ulcer of the left eye, without benefit.

On general examination, the entire body of the patient was observed to be covered with brownish scales. The palms and soles were blackish and deeply fissured. The skin of the face was taut, and there was severe ectropion of both lower lids with slight ectropion of both upper lids. Photophobia and epiphora were pronounced. Examination with the slit lamp showed that the cornea of the right eye was clear, while the cornea of the left eye showed scarring just below the pupillary area and active ulcerative keratitis on the temporal side.

It was obvious that to correct the ectropion, which was the basic cause of the exposure keratitis, a skin graft would be necessary. But as the skin of the whole body was not normal and no members of the family were available, a skin donor was obtained. Patient and donor were of the same blood type and were cross matched as to blood type and M, N and Rh factors.

On Jan. 18, 1948, with the use of tribromoethanol solution anesthesia, a median tarsorrhaphy was performed on the patient's left eyelids. A linear incision was made parallel to the lid margin, and the skin was freed on either side. A free graft, taken from the donor's thigh, was fitted into the area, which had been prepared with thromboplastin solution. The lids were then covered with oiled silk and a firm pressure

bandage applied. The first dressing was done ten days later, and the isograft was found to be adherent, pink and free of discharge.

About twenty days later a similar operation was performed on the right eyelids, using the same donor. Ten days later, on first dressing, the graft was found to be adherent, pink and healthy.

Ectropion in patients with ichthyosis congenita is not a constant feature. According to the cases reviewed from the literature, the presence or absence of ectropion depends on the presence of the following factors: (1) involvement of the face and (2) atrophy of the skin, as well as on the degree and severity of the atrophy. The skin of the face atrophies and the skin of the eyelid retracts, thus causing eversion of the lid margins. Ectropion of all four lids is extremely rare, occurring only in severe cases; in such cases the patient usually dies in the first year of life.

Cases similar to the one presented, and with successful isografting, were reported by Elschnig (*Klin. Monatsbl. f. Augenh.* **1:** 17, 1912) and by Shimkin (*Brit. J. Ophth.* **29:** 363 [July] 1945).

In a review of the American literature no previously reported case of this type could be found.

DISCUSSION

DR. JAMES S. SHIPMAN: This case brings to my mind one I saw about ten years ago in which this operation should have been done, and was not. Indeed, it would have been fortunate for the patient if she could have had such an operation earlier in life. The patient was a white woman about 40 years of age who had made her living for many years as the "alligator woman" in a carnival. She had an extreme ichthyosis, and there was no mistake about her skin's looking like that of an alligator.

Dr. Spaeth is to be complimented on the excellent result in his case. I had the pleasure of seeing this boy at Wills Hospital before and after his operation, and the result was all that one could ask for. However, I should not destroy for at least another six months the beautiful adhesions holding these lids together. The boy is able to see to get around, and I should be concerned about these lids turning out again if they are cut too soon. I do not like to disagree with Dr. Spaeth, who has had much more experience with these cases than I have, but I feel that one must wait a long time before cutting these adhesions.

Follow-Up Studies in Five Cases of Intraocular Nonmagnetic Foreign Bodies. DR. JAMES S. SHIPMAN.

Six cases of intraocular copper foreign bodies were presented. Nearly sixteen, fifteen and thirteen years, respectively, had elapsed since operation in the first 3 cases. In cases 4 and 5, it had been over three years since operation. In case 6 the operation was done only one week prior to this report, and the patient was still hospitalized. In 5 cases the copper body was in the vitreous, and in 1 case it was in the anterior chamber. The first, third and fifth cases were similar in that the injuries were all incurred from coil winding machines at the same plant and the foreign body was a fine piece of copper wire. In the second and sixth

cases the injury was due to the explosion of a dynamite cap, and in the fourth case, to the explosion of a .22 caliber cartridge.

A technic was described for the removal of nonmagnetic foreign bodies from the vitreous and the anterior chamber respectively.

In the first 4 cases, all with copper in the vitreous, a posterior sclerotomy was done, and through the lips of the wound a small capsule forceps was inserted; while looking with the ophthalmoscope through the dilated pupil, the operator was able to grasp the foreign body in the vitreous and extract it through the sclerotomy opening. In the first case the scleral opening was closed with interrupted black silk sutures, without cauterization. In the other 3 cases the same method was employed, but the sclera was coagulated around the incision with diathermy, before suturing.

In the first case recovery was uneventful and vision was normal for five weeks after the operation. At the end of this time a retinal detachment was observed. This became more pronounced, and approximately two months after the original accident the first operation for retinal detachment was done with the Weve needles. This procedure was unsuccessful, and two more operations were performed, Safar pins being employed in the last. These attempts were also unsuccessful. At the time of the report, more than fifteen years after the patient's injury, the injured eye had no light perception, presented a complicated cataract and was divergent.

In the second case recovery was uneventful, and although the patient had a partial posterior cortical cataract, from the time he was first seen until almost fifteen years after the injury, he had had useful vision, 6/15 with correction. There was never any evidence of retinal detachment, and the visual field had remained full.

In the third case a more intense reaction followed operation than in the first two cases. The partial cataract which was present before operation became complete shortly afterward, and a linear extraction was necessary. After the cataract extraction, the patient made an uneventful recovery and had had no trouble since. At the time of the report, thirteen years after the injury, the eye was entirely quiet, with normal tension. The vitreous was clear, and the fundus was healthy, with no evidence of detachment. The visual field was full, and visual acuity of the injured left eye was 6/9 + with a + 13.00 D. sphere.

In the fourth case the prognosis was poor, owing to the very large foreign body and the massive preretinal hemorrhage which was present when the patient was first seen. A considerable amount of vitreous had been lost, and further hemorrhage occurred at the time of extraction of the foreign body. This hemorrhage persisted, and eventually the eyeball became phthisical. Enucleation of this eye was advised.

In the fifth case the copper wire was present in the anterior chamber. The wire was removed by means of a paracentesis made with a keratome and enlarged with scissors. The foreign body was then grasped with iris forceps and withdrawn. However, the iris became entangled and had to be replaced through the wound. This resulted in some incarceration, but no prolapse of the iris in the paracentesis wound. The pupil was pear shaped and drawn to the nasal side. A localized opacity still remained on the nasal side of the lens capsule. The media

were otherwise clear; the fundus was healthy, and both eyes were entirely quiet. At the last report, visual acuity, without correction was 6/5 in the right eye and 6/6 in the left eye.

The sixth, and last, case reported was one in which the nonmagnetic foreign body was localized in the vitreous and the same procedure was followed as in the other cases of nonmagnetic foreign bodies in the vitreous. However, in this case the results were much better than in the others, owing to the fact that after the sclerotomy opening had been made a bead of vitreous presented with the piece of copper glistening in the center of the bead. This was then extracted with forceps and the necessity of going any deeper into the vitreous was obviated. The scleral wound and the conjunctiva were then closed in the same manner as in the other cases. At the time of this report this patient was still hospitalized, and the outcome was uncertain.

Comment.—It was later realized that in the first case the eye should have been saved. The retinal tear made by the posterior sclerotomy should have been sealed off, and the detachment could have been repaired. Failure to do this was the result of lack of sufficient knowledge, sixteen years ago, of the method of closing retinal tears. In cases 2, 3 and 5 the results were as satisfactory as one could expect with such drastic surgical intervention. The situation in case 4 was considered unfavorable because of the large size of the foreign body and the massive preretinal hemorrhage. However, a larger sclerotomy might have been done earlier. It was realized in retrospect that an iridectomy should have been done in the fifth case.

The author stated that in his opinion the results obtained in the 5 reported cases of copper in the vitreous justify the technic used. However, a special type of forceps, such as a modification of the alligator forceps used by otologists, or a smaller and stronger forceps similar to those of Thorpe's, might aid in grasping and holding the foreign body. If the lens is too opaque for one to see the foreign body, the procedure described cannot be employed. However, since removal of the cataractous lens would be indicated, in any event, the extraction could be done first, followed by the technic described.

DISCUSSION

DR. GEORGE H. CROSS: Dr. Shipman has been fortunate in having cases in which the intraocular foreign body could be seen with the ophthalmoscope. Usually the lens is cataractous or the vitreous is filled with blood or exudate, through which it is impossible to see the foreign body. In such a case the foreign body is best removed with the aid of a biplane fluoroscope. With this method the foreign body is accurately localized roentgenologically.

Dr. Shipman was fortunate in having most of the foreign bodies small ones. He spoke of the use of the endoscope, which to my mind requires a large opening in the eyeball.

My experience with nonmagnetic foreign bodies is limited to about 100 cases, 50 of which were treated surgically. In many cases the localization was poor, or the foreign body was outside the eyeball. In 1 case it was localized in the lower conjunctival cul-de-sac. It is essential to have good localization of the foreign body.

If one has a good roentgenologic report, but cannot see the foreign body, and knows that it is nonmagnetic, one may try the biplane fluoroscope.

Dr. Edmund B. Spaeth: Dr. Cross has done a tremendous amount of work with the biplane fluoroscope. I know so little about the use of this instrument that it is perhaps unseemly of me to discuss it. I am not at all convinced that the biplane fluoroscope is good for the removal of nonmagnetic foreign bodies. The trauma done to the eye because of the mechanical difficulties connected with the use of the forceps without visualization of the operative field is too extensive for approval.

I wish to speak in greater detail about the use of the endoscope. I have had a fair amount of experience with this instrument and have found it valuable in cases in which it can be employed. Of course, one cannot use the endoscope when the vitreous is full of hemorrhage.

There are many factors connected with the removal of nonmagnetic metallic foreign bodies from the eye, and many different means of localization and extraction. Each individual case may require an individualized technic. At best, these cases frequently present difficult problems and should be studied in detail as to the method most applicable to the situation present. Dr. Shipman is to be congratulated on his results in these cases. The real tragedies in ophthalmology are the cases in which the foreign body could not be removed. I believe that every eye with a retained intraocular foreign body except for those of zinc and aluminum will be lost ultimately. With some substances, such as lead shot, the initial injury is so great that the eye is destroyed irrevocably at the start.

Dr. James S. Shipman: With respect to the point that it was my good fortune that these foreign bodies could be seen, I do not feel that an opaque lens is a contraindication to this procedure. The lens must be removed anyway; then the foreign body can be seen and removed with the technic described.

I feel that, in order that a piece of copper be seen and removed by means of the biplane fluoroscope, the foreign body would have to be rather large, and in such a case the eye would be practically destroyed.

I doubt whether any of the foreign bodies in the cases I have described could have been seen well enough with the fluoroscope to warrant their removal by this method, since they were all unusually small pieces of copper.

Relation of Cerebrovascular Resistance and the Grade of Retinal Changes in Hypertension. Dr. Irving H. Leopold, Dr. Seymour S. Kety (by invitation), Dr. William A. Jeffers, Dr. Joseph H. Hafkenschiel (by invitation) and Dr. Henry A. Shenkin.

Twenty-one hypertensive and 3 nonhypertensive subjects were studied, in an effort to find whether or not there was any correlation between the retinal vascular changes and the cerebrovascular resistance associated with hypertension. The cerebral blood flow was determined by the method of Kety and Schmidt, and the retinal vascular changes were evaluated according to the classification of Wagener and co-workers.

It was found that a statistically significant correlation existed between retinal changes and the cerebrovascular resistance. The relation was a direct one in that as cerebrovascular resistance increased, the grade of retinopathy also tended to increase.

It is evident that the retinal changes do reflect with some accuracy the state of the cerebral circulation, but the degree of accuracy is not high, in that one cannot predict from the ophthalmoscopic findings the exact extent to which the cerebrovascular resistance has been raised.

Dr. Seymour S. Kety: When the physiologist studies hypertension in man, he is impressed with the fact that practically everything he investigates proves to be normal. The output of the heart is normal; the blood flow through the kidney is essentially normal except in the very late stages; the blood flow through the skin, the periphery of the body and the muscles has always been found to be physiologic. If he calculates the resistance in the blood vessels of these various organs, he finds a uniform increase in the peripheral resistance throughout the body and in any individual organ. Since the brain has been implicated in numerous etiologic theories of hypertension, we thought it of great interest to study the cerebral blood flow, and finally a method was developed at the University of Pennsylvania which permitted such a determination. It was found that exactly the same amount of blood passes through the brain of the hypertensive patient as passes through the brain of a normotensive person. The interesting thing, however, was that this occurred even though the blood pressure might be twice the normal. This must indicate a high degree of resistance in the blood vessels of the brain that keeps back the flow of blood which otherwise would be much above the normal.

Dr. Leopold has discussed the possibility which that finding presented to him as an ophthalmologist—that now, with a method for quantitating the resistance in the brain, one could evaluate the clinical impression of clinicians and ophthalmologists that the changes in the eyegrounds are a reflection of what is going on in the brain. We were gratified to find that there was a fairly good correlation between the two; that the correlation is not better is not surprising, for one must remember that we were measuring the vascular resistance at one time, at the time of observation, and that resistance was a function of the degree of spasticity of the vessels at that particular moment. The changes in the eyegrounds according to the classification used not only are a measure of the degree of spasm in the vessels at that particular moment but also reflect how long that spasm has been going on, and how severe it is. These changes in the eyegrounds are the result not only of spasm, but of spasm over a long period of time. If it were possible to separate the various factors that enter into retinopathy, particularly the degree of hypertonus at the time of observation and the results of the past history of that tonus, one might get an even better correlation than the quite satisfactory one that Dr. Leopold has found.

Dr. Francis Heed Adler: I should like to ask Dr. Leopold whether there is any difference in the cerebrovascular resistance in patients with atherosclerosis and that in patients with arteriolosclerosis. Dr. Leopold is evaluating the changes in the eyegrounds which admittedly are

severer in cases of arteriolosclerosis. It is possible that patients with atherosclerosis might have high cerebrovascular resistance and yet show no pathologic changes in the fundus. This state would upset the true correlation between cerebrovascular resistance and hypertensive retinopathy seen in cases of mild and rapidly progressive hypertensive vascular disease.

There are still many things which are not known about the retinal circulation. It has always been taught that the ocular vessels are end arteries, for they are said to be like the cerebral arteries. Until fairly recently, it was believed that the cerebral arteries were end arteries in the sense of Cohnheim. Lorente de Nó stated that the cerebral arteries are in no sense end arteries and that blood cells in the capillaries can pass freely from one end of the cerebral cortex to the other. It may be time that the retinal circulation be reinvestigated to determine whether there are anastomoses between the various branches of the retinal arterial tree.

DR. I. S. TASSMAN: It is difficult properly to digest and interpret in such a short time a presentation of this kind. A question occurs to me, especially with reference to the point that Dr. Adler introduced, "Is it possible to correlate findings of this kind for practical purposes?" The vascular beds in the two organs supplied, for example, are of different natures. There are differences in these structures, as well as possible differences in the arterioles of the brain and those of the retina.

In their origin, course and number, the cerebral vessels, with their ramifications, are quite different from the retinal vessels, so that a difference in the vascular resistance of the two sets of vessels might be expected. Is it possible, therefore, to correlate satisfactorily the resistance of the one with the pathologic changes in the other?

DR. IRVING H. LEOPOLD: The figures for normal cerebral blood flow are based mostly on values found in young persons between 20 and 30 years of age. The hypertensive patients studied here were also from a young age group, approximately 20 to 45, for the most part. In this group senile atherosclerosis would probably have a low incidence. The same cannot be said for local atherosclerosis. As yet no correlation of atherosclerotic change in retinal vessels with cerebrovascular resistance has been made.

The retinal and cerebral vessels differ in many ways. There is, for example, no cerebral counterpart of the crossing phenomenon of retinal vessels. Nevertheless, evidence has been presented in the past suggesting that the two systems of vessels show similar changes, and the present study also demonstrates a significant relation in the presence of hypertension.

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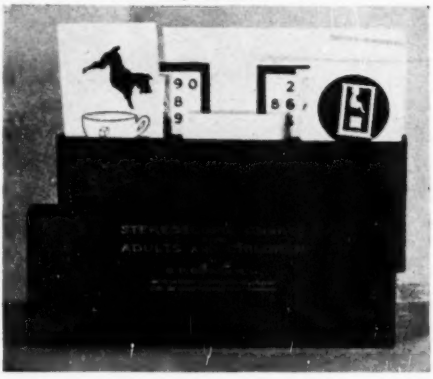

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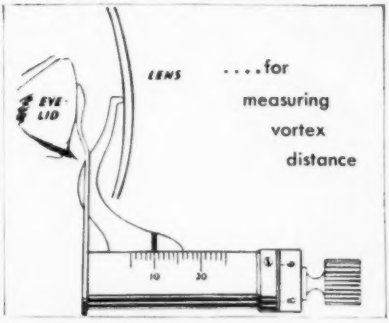

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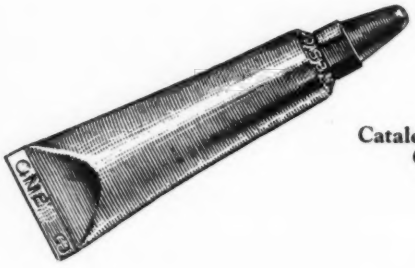

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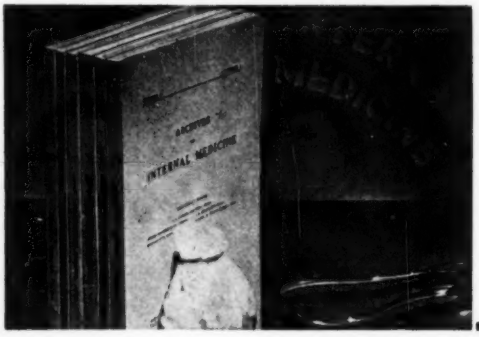

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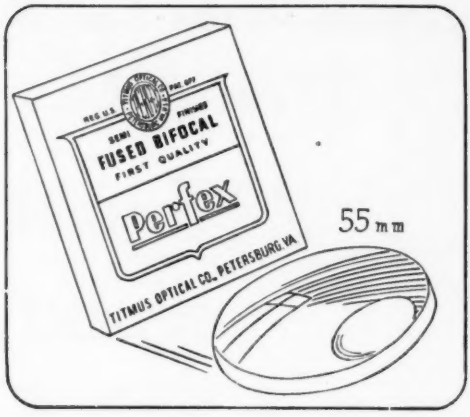

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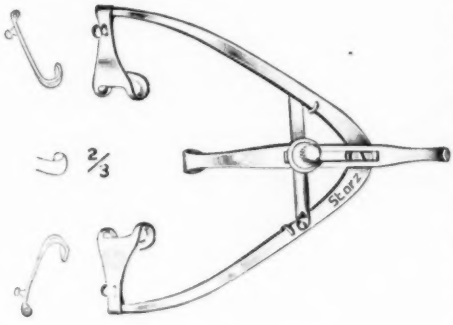

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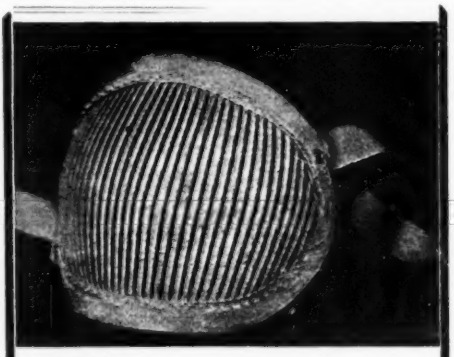

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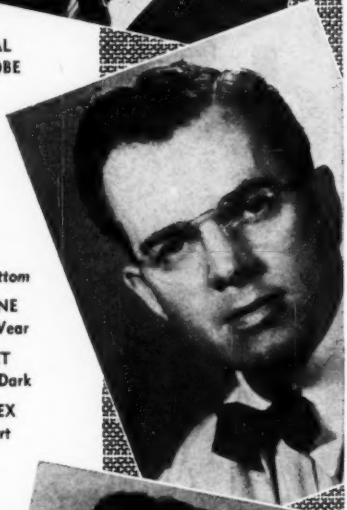

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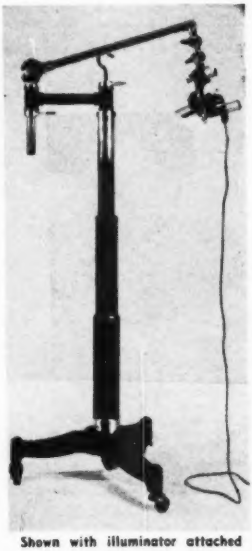

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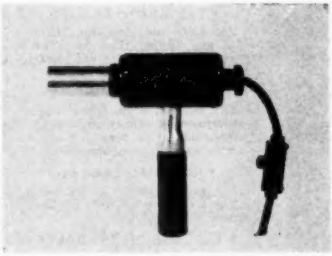

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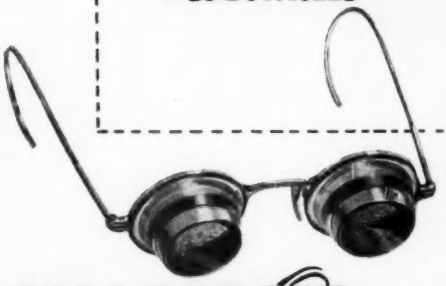

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